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ON GONORRHŒAL SEPTICÆMIA AND ENDOCARDITIS.[1]

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The comparative frequency of gonorrhœal septicæmia and endocarditis may be regarded as generally recognized, and, since the positive results obtained by Blumer,[2] Lazear,[3] and the writer,[4] abundant proof of the specific nature of the infection in many of these cases has been accumulated. At the same time the observations of many students have shown that a demonstrable septicæmia may exist in connection with various complications other than endocarditis.

While the communication of Gurvich[5] has made evident the fact that gonorrhœa may be followed by mild forms of valvular disease, those cases which have particularly attracted the attention of clinicians have been instances of grave malignant endocarditis. The clinical picture of these cases differs in no essential particular from that associated with ulcerative endocarditis from other causes.

The development of symptoms suggestive of septicæmia with fatal results, consecutive to gonorrhœa, without evidence *intra*

[1] Read before the Association of American Physicians, May, 1905.

[2] Thayer and Blumer. Endocardite ulcéreuse biennorrhagique. Septicémie d'origine biennorrhagique. Arch. de méd exper. et d'anat. pathol., 1895, vol. vii., p. 701; also, Johns Hopkins Hosp. Bull., Baltimore, 1896, vol. vii., p. 57.

[3] Thayer and Lazear. A second case of gonorrhœal septicæmia and ulcerative endocarditis with observations upon the cardiac complications of gonorrhœa. Jour. Exper. Med., New York, 1899, vol. iv., p. 81.

[4] Thayer. Gonorrhœal endocarditis and septicæmia. Compt. rend. cong. internat. de méd. 1897, Mosc., 1899, vol. iii., p. 5, sec. 350–367.

[5] Russk. Arch. Patol. klin. Med. i. Bakt., 1897, vol. iii., p. 329.

vitam of any focal complication, is also an occurrence of no great rarity. In most of these instances, however, necropsy reveals some local focus of infection outside of the urethra. Dr. Osler, for instance, tells me of a case of lethal septicæmia consecutive to gonorrhœa, which presented, during life, no localizing symptoms, where, at necropsy, the only focal lesion to be found was a prostatic abscess no larger than the end of the thumb. A similar case was under my care thirteen years ago, in which, however, no necropsy was obtained. A young man of twenty-two developed, in connection with an acute urethritis, an intermittent fever of extreme severity. Observed for several weeks in the hospital, the heart sounds were perfectly clear, and no evidence of any localization of the infection was to be detected beyond the history of a very slight arthritis, which had entirely cleared up. The patient died several weeks after leaving the hospital.

Padula,[1] in 1892, described a form of mild continued or intermittent fever which he had observed in a number of instances of gonorrhœa, sometimes in simple cases, but more particularly in those with complications. After running a course of several weeks' duration the fever disappeared.

From our present knowledge of the biological characteristics of the gonococcus it is natural to regard these cases, both mild and severe, as instances of true septicæmia. While this is well known to be the case in the more severe cases, the proof of the existence of septicæmia has not as yet been afforded in the milder instances.

It was, however, our good fortune last summer to observe an instance of continued fever of about seven weeks' duration occurring in a case of acute gonorrhœa, without apparent complications, in which the existence of a septicæmia was established by cultures from the blood. The case will be described later in this communication.

Our experience with gonorrhœal endocarditis and septicæmia in the Johns Hopkins Hospital has been rather interesting. Apart from the eight cases reported by Blumer,[2] Lazear,[3] Harris and Dabney,[4] Johnston,[5] and myself,[6] there have occurred two recent instances of ulcerative endocarditis of gonorrhœal origin, to which I shall refer later, making in all six cases in which gonococci were positively identified by cultures from the blood *intra vitam* (Thayer and Blumer), from the local lesions at necropsy (Harris and Dabney, Case I.; Thayer, Case II. of this communication),

[1] Febbre infettiva da virus blennorrhagico. Roma, 8vo., 1892.

[2] Op. cit.

[3] Op. cit.

[4] Report upon a case of gonorrhœal endocarditis, etc. Johns Hopkins Hosp. Bull., Baltimore, 1901, vol. xii., p. 68.

[5] Harris and Johnston. Gonorrhœal endocarditis, with cultivation of the specific organism from the blood during life. Johns Hopkins Hosp. Bull., Baltimore, 1902, vol. xiii., p. 236.

[6] Op. cit.

or by both methods (Thayer and Lazear, Harris and Johnson, and Thayer, Case III. of this communication).

In one case (Harris and Dabney, Case II.) the presence of characteristic organisms at necropsy, taken in connection with the absence of growths on ordinary media, afforded almost positive proof of the specific nature of the infection.

In three cases (Thayer and Lazear, Case XXXII. of the table, and Harris and Dabney, Cases III. and IV.), with an evident mixed infection, organisms were found at necropsy microscopically and tinctorially characteristic of gonococci. In one of these (Harris and Dabney, Case III.) diplococci, morphologically and tinctorially characteristic, were found in the pericardium, while cultures from the same source were negative. In the other two cases, although the cultures from the valves showed other organisms, the infection clearly followed an acute gonorrhœa without other apparent cause.

In addition to these instances I have, in the past six years, seen in consultation four cases in which an ulcerative endocarditis has followed gonorrhœa. In only one of these instances were cultures taken *intra vitam*, and here streptococci were obtained. In one other, in which the necropsy revealed an ulcerative mitral endocarditis, an unidentified bacillus was cultivated. In all of these cases, however, the connection of the urethritis with the development of the symptoms, was such as to make it probable that the acute infection had served as the portal of entry. Such an experience as this is sufficient to emphasize the fact that an acute urethritis is by no means infrequently followed by an endocarditis, either of specific gonorrhœal nature or due to secondary or mixed infections which have found their portal of entry in the urethritis or have settled later upon the primarily infected valves.

The last two hospital cases, which will be reported at the end of this communication, presented the usual features of ulcerative endocarditis—irregular, intermittent fever, chills, progressive anæmia, and nephritis—developing on the one hand in a case of acute gonorrhœa with arthritis about five weeks after onset, and in the other during a demonstrable urethritis, which the patient, a colored man, denied. It is interesting that in both of these cases, with a pure aortic lesion, there was a well marked, presystolic, rumbling (Flint) murmur, suggestive of mitral involvement. In one of the cases, indeed, a diagnosis of mitral disease was made. Cultures from the blood during life were positive in one, negative in the other. In both cases pure cultures of the gonococcus were obtained at necropsy from the affected valves.

The remaining case, which is regarded as of especial importance, is an instance of gonorrhœal septicæmia, with continued fever of seven weeks' duration.

CASE I.—*Male, aged twenty-eight years; gonorrhœa; posterior urethritis; continued fever of seven weeks' duration suggestive of typhoid; gonococci obtained in pure culture from the circulating blood; recovery.*

Hospital No. 47,439; Medical No. 17,342. X., aged twenty-eight years, was admitted to the Johns Hopkins Hospital on 17th July, 1904, complaining of headache, fever, and a general "run-down" condition.

Family History. Mother, living and well. One brother has some trouble with his stomach. One brother and one sister died in infancy. Family history, otherwise good.

Previous History. Has always been strong and well. As a child, had scarlet fever, chicken pox; thinks that he had typhoid fever at the age of six or seven. Eight years ago he had two sores on his penis which were said to be chancres. He was treated constitutionally and had no secondary symptoms. Later, he has been assured by physicians that the lesions were chancroidal. Four years ago he had an attack of gonorrhœa with posterior urethritis. Three months ago, had a second attack, which has continued active to the present time. Has been a moderate drinker until eighteen months ago, since when he has been abstemious.

Present Illness. On 10th July, on the first day of a vacation, while in the country, he was seized with a severe headache and fever; no chills. On the following day he was seen by Dr. S. Sydney Kellum, of Bellhaven, Virginia, who found that he had a temperature of 103.5° and was restless, complaining of severe pains in his head and back. He complained of having felt "weak and bad" for a week or so. The tongue was coated; the pulse about 90. The patient was given a laxative. On the following morning the temperature was 99.8°. He was up and about. R—Quin. sulph., gr. iv, every four hours. In the afternoon, however, his temperature was 102.6°. On the three following days the condition was about the same. On the evening of the 15th of July the temperature was 103.6°. The splenic dulness was slightly increased and there was some tenderness on pressure. On entering the hospital the patient had complained only of severe headache, fever and constipation. His mind was much taken up with the fear that he had lues.

18th July, 1904. Red blood corpuscles, 5,024,000 to the cubic millimetre; leucocytes, 9000 to the cubic millimetre; hæmoglobin, 92 per cent. Smears from the urethral discharge, which was fairly profuse, showed large numbers of gonococci.

19th July. On this date Dr. Cole noted that the patient was well nourished; face flushed; mental condition quite clear and bright. He moved himself in bed readily and did not appear very ill. The tongue had a thick yellow coat; pupils widely dilated. The chest was well formed; expansion good and equal; everywhere resonant. Auscultation, clear throughout.

Heart. Point of maximum impulse was seen and felt in the fourth interspace just inside the mammillary line. The dulness extended just to this point. There was no dulness to the right of the sternum. The heart sounds were everywhere clear and regular. Pulse regular, 23 to the quarter, of good force and volume; no dicrotism. The vessel wall was not thickened. The *abdomen* was full, though not distended. There was no abdominal pain; no tenderness. The edge of the *spleen* was felt about a finger's breadth below the costal margin, hard and not tender. Over the entire body, especially on the chest and thighs and also on the face, forearms and hands there was a diffuse slight mottling of the skin. At places these small bodies are slightly raised, particularly on the forearms; no characteristic rose spots. There was a slight urethral discharge.

20th July. On this date I saw the patient, noting that the spleen was distinctly palpable, well below the costal margin.

23d July. Leucocytes, 8900. The temperature had ranged daily between 99° and 103° with regular morning remissions. The blood was negative for

malarial parasites. On the evening of the 23d the temperature reached 103.8°, being affected little by sponging. After an ice sponge at 10 o'clock at night, however, the temperature fell to 98.4°, rising, however, soon afterward to 102° at 4 A.M. There was a slight chill on the afternoon of the 23d.

24th July. Dr. Cole noted that the lungs were clear throughout.

Heart. Impulse not seen or felt. Sounds best heard in the fourth and fifth spaces just inside the mammillary line; clear. Pulse, 27 to the quarter, of moderate volume and tension, a trifle irregular.

25th July. The temperature remained elevated, being 101.2° at eight in the morning. Fresh and stained blood was negative for malarial parasites. On this date Dr. Thayer noted that the patient was in good condition. The tongue had a pasty coat; the abdomen was natural.

Heart Apex impulse is not sharply defined; it is, however, felt just outside the mammillary line, in the fifth space, 12.5 cm. from the midsternal line. The dulness extends 0.5 cm. outside of this and 4.5 cm. to the right, the greatest diagonal dulness being 15 cm. The sounds, loudest at the point of maximum impulse, are clear excepting for a slight reduplication of the first sound which is rather better heard over the right ventricle. In the pulmonic area the second sound is sharp and clear, no reduplication. The sounds are also clear in the aortic and tricuspid areas.

On the 26th, the patient complained slightly of a sore throat, the pharynx being a little injected. The right tonsil was slightly swollen with two small white follicular plugs. The pulse was about 25 to the quarter, of good quality, regular, nothing new on examination of the heart. There were a few rather suspicious papules on the abdomen suggestive of rose spots although not wholly typical. There was no tenderness and no distention of the abdomen. Widal tests, made on the 21st and on this date, were entirely negative. A rectal examination showed that the prostate was small, soft and rather tender. There were no nodules in the seminal vesicles. There was a marked right varicocele, while the left epididymis appeared to be somewhat indurated near the globus minor. The temperature, which had been above 104° on the two preceding days, fell lower in the morning, rising only to 103.6° at night. On the 27th the general range was a little lower. The leucocytes were 9000 to the cubic millimetre.

28th July. It was noted by Dr. Thayer that the heart sounds were clear at the apex and base and in the tricuspid area. At the end of inspiration the first sound in the pulmonic area was a little prolonged.

The diagnosis throughout had been regarded as lying between a gonorrhœal septicæmia associated with an endocarditis, or typhoid fever. Despite the slight increase in leucocytes we had been growing more inclined to regard the case as one of typhoid fever. It was deemed wise, however, on this date, to take cultures from the circulating blood. These were accordingly made by Dr. Brush. 1.5 c.c. of blood were therefore withdrawn in the usual manner from the median basilic vein, from which two agar plates were made. The vein was then cut down upon and about 7.7 c.c. obtained from which one agar plate was made.

At 8 P.M. on this day the patient complained of pain in the lower præcordium, and to the right of the sternum. No friction rub, however, was heard.

29th July. The temperature still remained elevated, ranging between 99.6° and 102.8°. The spleen was readily palpable. The heart sounds were quite clear. There were several suspicious rose spots. Pulse, 22 to the quarter, regular, not dicrotic. During the next several days there was little change in the condition of the patient.

Results of cultures taken on 28th July. In twenty-four hours there was a diffuse growth of minute grayish colonies on the first two plates, those in which the smaller quantity of blood was used. In the third plate there were a very large number of colonies of *bacillus subtilis*, a contamination.

Subcultures on blood agar from the first two plates yielded a faint growth of discrete, pin-point, almost translucent colonies composed of a diplococcus, which was almost entirely decolorized by Gram. Transferred to agar there were, in twenty-four hours at 37°, many pin-point, discrete grayish colonies unchanged in forty-eight hours and twelve days. On the plain agar slant there were very delicate, pin-point, slightly elevated, translucent islands along the smear. No change in forty-eight hours or twelve days. There was no growth on glucose agar, potato, bouillon, or litmus milk. The colonies consisted of biscuit-shaped diplococci growing in pairs, tetrads and short chains. They were immobile and decolorized by Gram's method. The cultural as well as the morphological and tinctorial characteristics justified the diagnosis of gonococcus.

From this time on there was a gradual fall in temperature. On 3d August, the temperature at eight in the morning, was 100.5°; leucocytes 12,000 to the cubic millimetre. The patient had been fairly comfortable, though there was a regular evening exacerbation of the fever and often slight chilly sensations. There was some little headache. The urethral discharge had disappeared.

4th August. On this date, Dr. Cole noted that the pulse was of good quality, 24 to the quarter; the tongue was still coated; the spleen readily palpable; the heart sounds clear. During the 6th, 7th and 8th the temperature gradually fell. On the 9th the highest point reached was 99.6°. On this date Dr. Cole noted that the patient's general condition was excellent; the tongue was clean; the heart sounds were clear. For the two preceding days the patient had complained of slight pain in the lower abdomen, but there was little or no tenderness and no distention. Leucocytes, 10,000 to the cubic millimetre. From this time on the temperature remained below 100°, excepting for a slight exacerbation to 100.8° on the 19th and to 100.4° on the 24th.

On 28th August it was noted that the point of maximum cardiac impulse was in the fourth interspace, 9.25 cm. from the midsternal line, the dulness reaching about to that point and 3.5 cm. to the right at the fourth rib. There was a slight cardio-respiratory souffle at the apex and a very slight scratchy murmur along the left sternal margin and in the pulmonic area. Pulse, 26 to the quarter, frequently a little irregular.

From this time on the patient steadily improved. There remained, however, a slight urethral discharge.

16th September. On this date, the day before his discharge, it was noted that the patient's convalescence was uninterrupted; he was gaining rapidly in weight and strength. His color was good. There was slight dyspnœa on walking. The patient was, however, very nervous.

Heart Point of maximum impulse not clearly seen or felt. Sounds best heard in the fourth interspace 9.5 cm. to the left of the median line. Relative cardiac dulness extends just to this point and 6 cm. to the left in the third interspace. To the right the dulness extends 5 cm. at the fourth rib. The sounds are well heard, clear and of normal relative intensity at the apex and base. In the pulmonic area there is still a short, rather slight systolic murmur heard over an area a little larger than the bell of the stethoscope and not transmitted. The pulmonic second is well heard, not accentuated.

17th September. The patient left the hospital in excellent general condition.

The *urine*, repeatedly examined during the patient's stay in the hospital, showed nothing remarkable, excepting a sediment containing at first much pus, which, however, disappeared in great part later. On several occasions there was a slight trace of albumin.

In response to a letter the patient called to see me on 11th February, 1905. He stated that he had been perfectly well since discharge excepting for the fact that he was still under Dr. Young's treatment for his posterior urethritis. He looked perfectly well. The pulse at the beginning of examination was 22 to the quarter, of fair size and duration, not remarkable in quality. The

maximum blood pressure by the Cook instrument with the broad band was 124; minimum about 108.

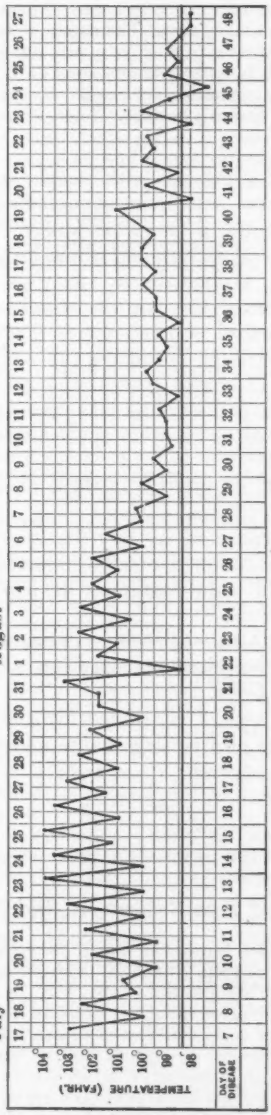


FIG. 1

CASE I.—Gonorrhœal septicæmia.

Heart. Point of maximum impulse, in the fifth space, a trifle inside the mammillary line. Relative dulness at the third rib; extends 2.7 cm. to the right of the midsternum and just outside the point of maximum impulse. Maximum diagonal dulness about 14.5 cm. The first sound at the apex is perfectly clear; second sound, clear. The sounds were also clear at the tricuspid pulmonic and aortic areas excepting for a very soft systolic souffle heard only at the base, louder in the pulmonic area, and an occasional reduplication of the second sound with inspiration, which was more marked on deep breathing. The heart's action is perfectly regular. There is no evidence of any cardiac lesion.

Here, then, was a patient with vague, general symptoms of headache, backache, and general malaise, continued fever, a thickly coated tongue, a palpable spleen, and suspicious rose spots. These general symptoms, which lasted seven weeks, were not unsuggestive of typhoid fever. In view, however, of the cultures and the course of the case the condition was clearly due to gonorrhœal septicæmia. Our suspicion throughout was that there might be an endocarditis, but at the subsequent examination, five months after his discharge, there was no evidence of cardiac involvement. If an endocarditis was present it must have been of so slight a degree as to leave no appreciable permanent damage.

The main points of interest in these observations would seem to be:

1. The demonstration of the fact that the mild, continued fever sometimes seen in connection with gonorrhœa without apparent complications is, in some instances at least, evidence of a true gonorrhœal septicæmia.
2. A true gonorrhœal septicæmia in cases in which there is no evidence of local complications may run a course not dissimilar to that of typhoid fever.
3. In doubtful cases of continued fever associated with gonorrhœa the possibility of a gonorrhœal septicæmia should always be borne in mind.

* * *

CASES OF ULCERATIVE ENDOCARDITIS FOLLOWING GONORRHŒA.

A. Two Instances of Pure Gonorrhœal Endocarditis with Autopsy.

CASE II.—*Male, aged twenty-one years; gonorrhœa; septicæmia; ulcerative endocarditis of the aortic valve with Flint murmur; death; autopsy.*

Hospital No. 48,901; Medical No. 17,794. C. L., colored, male, aged twenty-one years, cart driver, entered the Johns Hopkins Hospital on 14th November, 1904, complaining of a cold, shortness of breath and cough.

Family History. Parents living and well. One brother and one sister, living and well. No family history of tuberculosis.

Previous History. Patient has always been strong and well. Denies acute infections including venereal disease. Neither drinks nor smokes. Has always done manual labor. Has been married sixteen months; wife is well.

Present Illness. Three weeks prior to entry he was exposed to a rain storm, working all day in his wet clothes and suffering from chilly sensations. On the following day he had a "cold" and a slight hacking cough for which he took medicine for two weeks without benefit. By the end of the second week (6th November) he noticed shortness of breath after coughing and on exertion, particularly on walking fast or going up stairs. There was considerable expectoration of "phlegm." On the morning of admission he noticed, for the first time, œdema of his feet and legs. There has been nausea and vomiting on several occasions. On the day of entry the following note was made by the house physician, Dr. Giffen: "The patient is a very well developed young man. Pupils are equal; tongue slightly coated; mucous membranes of good color. Superficial glands of the neck are palpable but not markedly enlarged. There is marked sclerosis of the veins of the arm.

"The thorax is well formed; angle wide; expansion equal. Vocal fremitus diminished over the left upper lobe; percussion clear throughout. Crackling râles are heard just above and below the right clavicle, as well as a few in the upper part of the right interscapular space and at both apices.

"*Heart.* Point of maximum impulse in the sixth interspace, 8 cm. from the midsternal line. Relative dulness extends 10 cm. to the left at the apex, 4 cm. to the right of the median line. Pulsation in the temporal artery is visible; presystolic venous wave in the neck; slight precordial pulsation. On palpation there is an impulse with both systole and diastole. The pulse is definitely collapsing, of small volume and pressure, 28 to the quarter. The vessel wall is sclerotic. On auscultation at the apex there is a resonant, rumbling diastolic murmur followed by a sharp first sound. The second sound is indistinct. At the base there is a rough diastolic, heard in the second right interspace, loudest in the third left interspace. The two sounds are very sharp and snapping, the pulmonic much accentuated. A diastolic murmur is heard over the body of the heart.

"The abdomen is slightly full. Relative hepatic dulness begins at the sixth right rib and extends three fingers' breadth below the costal margin in the mammillary line. The spleen is not palpable. There is œdema of the feet and slight œdema of the legs; genitalia normal."

Blood. Red blood corpuscles, 4,480,000 to the cubic millimetre; colorless corpuscles, 9700 to the cubic millimetre; hæmoglobin, 90 per cent.

The temperature, which, on entrance, was 98.6° fell by 8 P.M. to 98.2° and to 95.6° at 8 the following morning. At noon on 16th November it was 96.2°.

16*th November.* At 9 o'clock the following note was made by Dr. Thayer: "The patient is propped up in bed. There is well marked throbbing of the carotids. The tongue has a white coat. The pulse is regular, 27 to the quarter, of moderate size, abrupt, suggestive of a Corrigan pulse, though by no means typical."

"Thorax, symmetrical; costal angle of about 90°; precordial region a little fuller than on the right side. Point of maximum impulse visible in the sixth interspace 9.7 cm. from the midsternal line. Impulse, not particularly strong. Relative dulness extends just beyond the point of maximum impulse and 3.8 cm. to the right of the midsternal line. The maximum diameter of the area of dulness is 15.5 cm. At the apex there is a distinct thrill felt best in the sixth interspace inside the point of maximum impulse and leading up to the impulse. At the apex a rumbling, presystolic murmur is heard ending in the first sound which, though feeble, is abrupt. The second sound is replaced by a soft, distinct, diastolic murmur. Immediately above and outside the impulse the diastolic murmur becomes more audible, and in the axilla, where the presystolic murmur has wholly disappeared, a dull humming first sound and a loud diastolic murmur are clearly heard. Passing inward in the sixth space the first sound becomes louder and the rumble is distinct; the diastolic murmur is feeble. Above the sixth rib, within the parasternal line, the pre-

systolic murmur disappears and there is heard a fairly sharp first sound and a distinct second sound with a loud aortic diastolic murmur. In the second and third left interspaces the first sound is dull and humming, the second sound intensely accentuated. In the second right interspace there is a faint systolic and a distinct diastolic murmur. An element of the second sound is, however, heard. The diastolic murmur is best heard in the fourth left interspace. In the carotids there is a faint systolic hum; in the femorals, a distinct, short systolic tone.[1]

"*Lungs.* There are still a few râles at both apices and an explosion of fine rales on slight cough at the right apex. When propped up in bed the liver is not palpable. By percussion, it extends apparently just below the costal margin in the mammillary line."

Examination of the fresh blood shows no malarial parasites. Leucocytes, 24,000 to the cubic millimetre.

18*th November.* The temperature which was normal on the morning of the 16th, rose in the afternoon to 102° and fell again to a subnormal point by the following noon, rising, however, again in the afternoon. On the morning of the 18th it was 95.7°; by noon, 102.2°. A note made shortly after this by Dr. Cole, states that the patient was very ill; respirations 10 to the quarter; pulse 28 to the quarter, forcible, of large volume—a typical Corrigan pulse. The cardiac impulse was 10 cm. from the midsternal line. There was a slight presystolic thrill. The shock of both sounds was well felt. The first sound at the apex was loud and snapping and followed by a very short systolic murmur. It was preceded by a very loud rumble heard throughout almost the whole of diastole and running into the first sound. This disappeared as one passed outward, and a soft, blowing, diastolic murmur was heard throughout the whole period. Over the body of the heart both sounds were loud and booming. A soft to-and-fro murmur was heard along the left sternal margin, the diastolic being louder than the systolic. Along the right sternal margin, at the base of the heart, these murmurs were superficial, and over the sternum at the level of the second intercostal space, there was a scratching sound almost suggestive of friction. In the first and second right and left interspaces and over the subclavians there was a rather coarse systolic murmur differing in quality from that heard elsewhere. Leucocytes, 22,500 to the cubic millimetre.

19*th November.* The intermittent fever still continued. The following note was made by Dr. Thayer: "The patient looks very ill, the face is puffy, the pulse larger than formerly, 21 to the quarter, definitely collapsing; there is no great change in the position of the apex impulse.

"*Heart.* The first sound is fairly sharp, and the diastolic murmur is succeeded by a long rumble followed by a slight first sound. As one passes above the fourth rib there is a slight systolic and diastolic murmur associated with an intensely accentuated second sound, most marked in the third left space. The diastolic murmur is loudest at the fourth cartilage. In the aortic area there is a short to-and-fro murmur. The dulness extends 4 cm. to the right of the midsternal line."

The irregular temperature still continued and two days later, on the 21st, it was noted by Dr. Thayer that the pulse seemed larger than formerly. "A little inside the nipple the first sound is rather abrupt and loud and the mid-diastolic rumble particularly intense. At the parasternal line the mid-diastolic rumble becomes feeble, the second sound sharper, and the soft early diastolic more audible. The accentuation of the second pulmonic sound is extraordinary." A tracing from the radial pulse was suggestive of aortic insufficiency, the pulse at this time being 36 to the minute and the maximum pressure (Riva-Rocci, narrow band) 103 mm.

Irregular, intermittent fever still continued, the temperature ranging between

[1] Ventricular systole.

97° and 103°. On the 23d the following note was made by Dr. Thayer: "The pulse is much larger than formerly and definitely collapsing. The point of maximum impulse is in the sixth space, 10.8 cm. from the midsternal line. There is a well marked thrill at the apex which is associated with the impulse, whether diastolic or systolic, it is difficult to tell by palpation. The rumble is clearly presystolic. The first sound is very feeble. Inside the apex there is a rumble leading up to a fairly sharp first sound. As one passes inward a soft systolic murmur becomes audible in addition to the diastolic. There is a to-and-fro murmur in the aortic area and along the left sternal border. The vessels of the neck pulsate markedly. The patient was distinctly losing ground."

On examination of the urethra a purulent discharge was discovered, the smears showing many pus cells and a moderate number of intracellular diplococci decolorizing by the method of Gram.

On the following day Dr. Cole noted that the patient was much worse. The respirations were 64, gasping; pulse 30 to the quarter, of Corrigan quality; marked throbbing of the vessels of the neck. The resonance over the left front was impaired, numerous medium and coarse râles being audible, while at the apex, the breath sounds were harsh with many fine crackling

FIG. 2

CASE II.—Radial pulse, 21st November, 1904.

râles and prolonged expiration. High up in the axilla the breath sounds were tubular, and numerous coarse râles were heard in both backs. There was no great change in the cardiac signs. During the day the patient became very noisy and the pulse was extremely rapid. In the evening stupor came on, followed by death at half-past 1 in the morning of 25th November.

The *urine* was free from albumin throughout, the specific gravity varying from 1012 to 1025. There were, however, occasional hyaline and granular casts and a varying number of leucocytes in the sediment.

The blood pressure, taken by the Riva-Rocci apparatus with the old, narrow band, was extremely low, the records ranging from 110 to 96 on the day of death.

On 18th and 21st November cultures were taken from the circulating blood. On the first occasion, 7 c.c. of blood were distributed upon one plate and two flasks. On the second occasion 20 c.c. of blood were distributed among three plates and three flasks. The cultures were negative.

In view of the discovery of the urethritis the diagnosis was gonorrhœal endocarditis. The remarkable accentuation of the second pulmonic sound, together with the loud, rumbling presystolic murmur and the rather abrupt first sound led us to a diagnosis of a combined mitral and aortic lesion. The necropsy, however, revealed a pure aortic endocarditis.

Necropsy (No. 2424) by Dr. MacCallum, nine hours after death.

Anatomical diagnosis: Acute vegetative and ulcerative endocarditis; bronchopneumonia; œdema of the lungs; parenchymatous degeneration of the kidneys; infarctions in the kidneys and papillary muscles of the heart; cardiac hyper-

trophy and dilatation; epicardial fibroid nodules; beginning arterio-sclerosis; ecchymoses in the mucosa of the bladder.

The *heart* is greatly enlarged; the enlargement of the left ventricle being especially prominent. The arteries are covered with complete chains of epicardial nodules. There is a thick, irregular, tendinous patch over the front of the right ventricle. The surface of the heart is, in general, smooth except for the epicardial nodules. The fat over the heart is gelatinous and dark yellow. The apex of the left ventricle projects at least 3 cm. beyond that of the right and constitutes the point of the heart. The right auricle is distended with post mortem clot. Its lining is smooth. The auricular appendage also contains a post mortem clot. The tricuspid and pulmonic valves are normal in appearance. The blood clot is very firm and makes a perfectly distinct mould of the valve. The mitral valve is normal in appearance. The chordæ tendineæ are delicate. The papillary muscles of the anterior leaflet show, however, an opaque patch of yellowish color bounded by a zone of dark red. On section this appears to be a necrotic, coagulated area surrounded by a zone of hemorrhage. On opening the aorta it is found that the edges of the valves are all delicate. The right coronary segment and the middle segment are normal throughout. The left coronary segment, however, has suffered a great change. The edge of the valve throughout is quite delicate. The base of the valve, however, is covered with large vegetations, and an extensive erosion of the tissue has taken place, so that the whole floor of the sinus of Valsalva is destroyed and the scissors can be passed from the left ventricle into the aorta through the open floor of the left coronary sinus of Valsalva. The valve thus remains as a bridge across the aorta from the lower edge of which there hang large irregular vegetations. The sinus is covered everywhere by rough vegetations. The remains of its floor project outward and are roughened in the same way. The sinus has been extended by ulceration into the wall of the left ventricle, especially anteriorly and toward the left where a cavity has been hollowed out in the upper portion of the wall of the left ventricle just behind the anterior mitral segment. The chief mass of vegetation which hangs in the blood stream and is covered with blood clot, measures about 18 mm. in length. It is soft and friable and has a folded, irregular, yellow surface. The post mortem clot may be pulled off from it like a glove finger leaving it quite clean and uniformly grayish yellow. The apices of all of the mitral papillary muscles are crowned by areas of necrosis. The heart muscle is fairly firm and the wall of the left ventricle measures about 2 cm. in thickness. On tangential section the muscle is very opaque and much mottled with pale grayish and dark red areas. The coronary arteries show no marked sclerosis. There is a uniform roughening of the intima, especially in those areas where the epicardial nodules have developed. The endocardium is elsewhere smooth. That of the left ventricle has spots of opacity shining through. The right ventricle measures about 4.5 mm. in thickness and about 8.5 cm. in length. The left ventricle measures 9.5 cm. in length. The aorta shows streaks of sclerosis not very far advanced.

Lungs. There is a small amount of fluid in the left pleura. On removing the left lung it is found to be non-adherent. The surface of the lung is slightly roughened by a few tags representing old adhesions. The pleural surface is everywhere smooth and glistening, but very opaque and grayish as if the pleura were greatly thickened. The apical portion of the lung is inflated and soft. The remainder of the upper lobe and the whole of the lower lobe are very heavy and firm to the touch. Along the edges of the lobes, particularly along the edge of the lower lobe there are numerous small pin-head sized nodules, which are grayish and somewhat translucent. These seem to be solid masses, and not merely air bubbles. The lymph glands at the hilum are somewhat enlarged. They are soft, reddened and very œdematous. The bronchi contain a certain quantity of frothy fluid and their walls are smooth. On section the

lung is very firm. The upper portion of the upper lobe is evidently air-containing. The lower portion of the upper lobe is quite smooth on section, of a grayish red color with abundant areas of darker gray or black pigmentation, and the tissue is relatively airless. The same condition is found throughout the lower lobe but not quite so uniformly, there being patches of air-containing lung scattered here and there. The consolidation involves, irregularly, the whole lower portion of the upper lobe and the whole of the lower lobe with the exception of the small scattered air-containing spaces or areas described. The right lung is quite densely adherent over the upper lobe; it is rather smaller than the left and is very much roughened over the upper and middle lobes by adhesions. There is a deep scar causing a depression in the upper lobe. The middle lobe is densely adherent to the upper lobe and also to the lower. The anterior portions of the lung are air-containing and crepitant. The posterior and lower portions of the middle lobe feel solid. The whole posterior portions of both the upper and lower lobes feel firm. The bronchi contain a blackish, frothy fluid. On section, the same type of smooth elastic consolidation is seen throughout the upper and lower lobes as described in the left lung. Between the upper and middle lobe there is a mass of dense white scar tissue which occludes the bronchi and vessels. The posterior part of the lung is very œdematous and very elastic and firm, dark, reddish gray in color and almost entirely airless. The cut surface is quite smooth. A bit of the lung just floats in water.

The *spleen* measures 14 x 9 x 6 cm.; weight, 200 gm. The capsule is roughened by old adhesions. On section the splenic pulp is somewhat swollen. The Malpighian bodies are very large and prominent, opaque, grayish white in color. The *stomach* and *duodenum* and *pancreas* are not remarkable. The *liver* measures 29 x 19 x 13 cm. and weighs 2450 gm. Its capsule is smooth and uniform in appearance. On section the lobulation of the liver is very distinct. The lobules are extremely large and have a yellowish-gray periphery and a reddish centre. The red portion is very sharply marked off from the grayer portion and frequently shows central points of orange yellow.

Kidneys. The left kidney is firm and not enlarged, measuring 11 x 6 x 5 cm. The capsule splits in peeling off and leaves a rather smooth surface which is of a grayish red color. On the surface in the middle line there appears a small opaque area surrounded by a dark red area. On section this opacity extends 4 mm. into the substance of the kidney and is surrounded completely by a zone of hæmorrhage. The cortex of the kidney is thick, measuring 12 mm. The striations are straight. The glomeruli are prominent. The epithelial portions of the cortex are rather opaque and gray. The right kidney presents no opacities on the surface. The capsule strips off smoothly. On section it presents the same general appearance as the left, the cortex being swollen and the glomeruli particularly prominent. The kidneys weigh 450 gm. together. The *adrenals* are apparently normal. The *cervical lymph glands* are much enlarged, very œdematous and dark red. The *thyroids* and *œsophagus* are not remarkable.

Bladder. The mucosa of the bladder is smooth. There are some ecchymoses at the fundus. The *urethra* is quite normal in appearance. The *testicles* and *epididymes* are normal in appearance.

Bacteriological Report by W. W. Waite. Cover-slip preparations were made from vegetations on the aortic valve and from the urethra. Plate cultures on ordinary media were made from the pericardial cavity, heart's blood, vegetations on the aortic valve, gall-bladder, liver, spleen and kidney. Rabbit's blood agar tubes were made from vegetations on the aortic valve.

Cover-slip preparations from the urethra stained with methylene blue showed several kinds of organisms. That present in greatest numbers was a diplococcus of a distinct biscuit-shape, which was inside and about pus cells. The cover-slip preparations from the vegetations showed in any single field

numbers of organisms which were all cocci arranged singly and in pairs. The paired organisms had their long axes parallel and were distinctly biscuit-shaped. None of the organisms held their stain when treated by Gram's method.

On the rabbit's blood agar tubes from the vegetations there were numerous small translucent colonies which appeared to be all of one kind. A gentian violet stain showed but one type of organism which was a diplococcus arranged singly and in pairs, which decolorized completely by Gram's method. Transfers were made to blood serum, plain slant agar and hydrocele agar. The blood serum and agar showed no growth. The hydrocele agar, however, contained many small clear translucent colonies on the surface of the medium. A culture allowed to grow twenty-four hours in the incubator at 36° C. and then kept at room temperature lived only two days.

Diagnosis: *micrococcus gonorrhœæ.*

The plates on ordinary media contained a few colonies of micrococcus roseus and micrococcus cereus. These were probably air contaminations or end infections. The heart plates contained a few colonies, the pericardium, 1; vegetations, 2; gall-bladder, 4; liver, 6; spleen, 22.

CASE III.—*Male; aged forty-two years; gonorrhœa; arthritis; septicæmia; ulcerative aortic endocarditis; death.*

Hospital No. 49,214; Medical No. 17,889. R. W., aged forty-two years, married, a dyer by trade, was admitted to the Johns Hopkins Hospital on 12th December, 1904.

Family History. Father, living and well. Mother, died at fifty-two of typhoid fever. Two brothers, living and well. Three brothers died in infancy. One sister, living and well; two died of unknown cause. Family history, otherwise good.

Previous History. Has always been a healthy man. Had a severe attack of scarlet fever at five, since when he has been bald. Measles at seventeen; whooping-cough at seventeen. No other serious illnesses. Seven years ago he suffered from "indigestion" for six months, but has had no trouble since then. Denies lues.

Habits. Drinks an occasional glass of beer; smokes one package of tobacco a week. Average weight, 148 pounds.

Present Illness. Four weeks ago developed an acute gonorrhœal urethritis. Two weeks ago noticed that his right wrist was tender and lame, so much so that he was obliged to stop work on the following day; it has been gradually growing stiffer and more painful. Three days ago his right shoulder became lame and for two days his right eye has been a little sore early in the morning. There has been no trouble with vision. Has not slept well for the last four nights, and for the last two nights there has been profuse sweating. There has been no cough; no expectoration.

On entrance it was noted that the patient was well nourished; tongue fairly clean; mucous membranes of good color. Slight conjunctivitis of the right eye. The pupils were unequal, the left the larger. Both reacted to light and accommodation. There was marked pulsation of the vessels of the neck and of the brachials. The skin was hot and dry. The lungs were clear throughout.

Heart. Point of maximum impulse in the fifth interspace 9.5 cm. to the left of the median line. Relative cardiac dulness begins at the upper border of the third rib, extending, in the fourth interspace, 2.5 cm. to the right and 9.5 cm. to the left. All over the heart is heard a soft, systolic bruit. Sounds otherwise clear. The second pulmonic is not accentuated, though there is a reduplication of the second sound in this area. Pulse, 21 to the quarter, regular in force and rhythm, volume good, tension good, vessel wall not felt. Blood pressure (Riva-Rocci, broad band), 125 mm. Abdomen natural, no tenderness. Neither spleen nor liver palpable.

Genitalia. Slight œdema with glairiness of the meatus. There was a small accessory meatus with a slightly glairy discharge as well. Gonococci were demonstrated in the discharge. No swelling of the epididymis.

Joints. Right wrist thickened, hotter than the other wrist, not especially painful nor tender, slightly reddened. Motion limited in all directions. Pain and limitation of motion in the carpo-metacarpal joints of the thumb. Other joints appear to be free.

Examination of the blood showed that the red corpuscles were regular in outline, of good shape and size. There was a slight leucocytosis but no parasites.

Blood Count. Red blood corpuscles, 4,960,000 to the cubic millimetre; colorless corpuscles, 10,600 to the cubic millimetre; hæmoglobin (Gowers), 98 per cent.

13*th December.* The temperature, 99° on admission, fell to 98° at 8 P.M. and was normal on the following morning. Note by Dr. Cole: "Slight injection of the conjunctiva; no purulent discharge; no opacity of the cornea; pupils equal; no glandular enlargement excepting for two glands about as large as peas in the left axilla, and the right epitrochlear gland, which is about the size of a pea. The left epitrochlear is just felt. Chest, well formed; lungs everywhere hyperresonant on percussion. Breath sounds distinct. A few piping rales.

"*Heart.* Point of maximum impulse not well seen nor felt. No absolute cardiac dulness. Relative cardiàc dulness extends 8.5 cm. to the left; above to the third rib. Slight reduplication of the second sound at the apex. Soft systolic murmur in the pulmonic area. Aortic and pulmonic seconds of normal intensity. Spleen not palpable.

"*Joints.* Complains of tenderness in the right shoulder. There is no swelling, no heat. The right wrist is swollen for the most part on its inner surface; temperature, elevated; moderate tenderness and swelling being most marked on the radial side of the dorsum of the wrist and about the head of the radius; do not extend up the sheaths of the tendons. No other joints affected."

16*th December.* The temperature remains normal. No great change in the symptoms. Dr. Osler noted on this date that there was a well marked systolic murmur.

18*th December.* Dr. Cole observed on this date that the right wrist was swollen, especially over the inner portion, the skin red and hot. The patient complained of throbbing pain in the joint, worse at night. The pain was sufficient to awaken him. There was no restriction of motion in the right shoulder; no tenderness. There was an appearance of atrophy of the interossei on the dorsum of the right hand. The motion of the wrist was restricted. There was œdema about the joint, which extended about one-third of the distance up the arm.

"19*th December.* Red blood corpuscles, 5,122,000 to the cubic millimetre; colorless corpuscles, 10,400 to the cubic millimetre; hæmoglobin (Gowers), 97 per cent.

"Urethral discharge still shows diplococci, which decolorize by Gram. The pain in the wrist continues. The swelling now extends half way up the arm, the redness and tenderness being most marked about two and one-half inches above the wrist, where there is a brawny œdema, the skin being red and hot. The wrist is stiff but not extremely tender. The heart sounds are quite clear (Cole)."

3*d January*, 1905. The patient has been in the hospital since 12th December and has had at intervals a small amount of fever reaching above 100°. For the past five days the temperature has been lower than usual. Yesterday morning it rose to 99.5°. The patient was out of bed at this time and complained only of a slight headache. This morning he arose as usual, feeling well, but was not especially hungry. At 8 A.M. the temperature was found to

be 100.5° and the patient was at once put to bed. The temperature steadily rose to 103.8° at 4 P.M. On examination, the tongue was found to be dry and red, but not coated. The pulse was 120; the lungs were clear throughout on auscultation and percussion. There was a soft, blowing systolic murmur heard, especially, along the left sternal border. The abdomen showed nothing remarkable. The spleen was not palpable. The patient complained of feeling drowsy and sleepy. Colorless corpuscles, 16,500.

On the succeeding day (4th January) Dr. Cole made the following note: "With a sudden rise of temperature which occurred yesterday there have been no symptoms excepting that the patient has complained of slight chilly sensations. The face is a little flushed, the tongue moderately coated and dry. The pharynx is red and injected though the patient complains of no soreness. Scattered over the abdomen, back, lower chest and also, to a lesser extent, on the arms, are small elevated red spots which disappear on pressure, and resemble somewhat rose spots except for their size which is 5 to 6 mm. in diameter. They are a little more elevated and surrounded by a zone of slight hyperæmia. The patient thinks that he has previously noticed such an eruption over the body. Respiration, quiet and slow; lungs, quite clear throughout on percussion and auscultation.

"*Heart.* Point of maximum impulse not seen nor felt. Relative cardiac dulness difficult to outline; extends apparently 9 cm. to the left at the fifth rib, to the upper border of the third rib above, and 2.5 cm. to the right at the fourth interspace. No impulse is to be felt anywhere over the cardiac area. At the apex both sounds are well heard, the second almost as loudly as the first, while the systolic and diastolic pauses are of almost equal length. The heart's action is not rapid and is regular. No murmur can be heard at the apex or over the body of the heart. Both second sounds at the base are snapping, the aortic second being louder than the pulmonic. Over the sternum and in the second and third left interspaces near the costal margin, the second sound is a little prolonged and, though no definite murmur can be made out, it may be that a faint diastolic souffle is present. The abdomen is full; neither tender nor distended. The spleen cannot be felt. The edge of the liver is palpable one finger's breadth below the costal margin in the mammillary line. The right wrist is still stiff. The patient also complains of tenderness in the left wrist, though no swelling or redness is to be seen." Cultures were taken on this date from the blood. 20 c.c. of blood were distributed on two plates of hydrocele fluid agar; four plates of plain agar; one flask of bouillon; one flask of litmus milk.

On the following day the colorless corpuscles were 8000 to the cubic millimetre. Blood pressure, 116 mm. The temperature was lower—not above 100°.

6th January. The sputum, small in amount, mucopurulent and tenacious, showed microscopically leucocytes and epithelial cells; it was negative for tubercle bacilli. The blood pressure at 5.50 P.M. was 135 mm.

There was no growth in the cultures made on 4th January.

7th January. The temperature on the 6th ranged between 100.2° and 102.5°. On the 7th it fell gradually to 99.6° at 8 P.M. Colorless corpuscles, 18,900 to the cubic millimetre.

9th January. On this date, Dr. Cole noted that the patient's temperature was lower; pulse 23 to the quarter, regular. There was pain and tenderness over the left side of the larynx; no swelling or redness. The eruption of reddish papules over the body had disappeared to a certain extent. The spleen could not be felt. The œdema and tenderness of the right wrist had almost entirely disappeared, but there was still some stiffness. The patient complained of a slight aching in his ankles.

Blood Count. Red blood corpuscles, 5,252,000 to the cubic millimetre; colorless corpuscles, 11,360 to the cubic millimetre; hæmoglobin (Gowers), 82 per cent.

On the following day (10th January) Dr. Cole noted that the papular eruption was disappearing; pulse, 26 to the quarter, regular, of rather large volume with a forcible quick impulse and a slightly collapsing quality.

The impulse of the heart was neither seen nor felt. Sounds, best heard in the fifth space 8.5 cm. from the midsternal line, to which point the relative cardiac dulness extended; to the right it reached 4 cm. in the fourth interspace, and above to the upper margin of the third rib. There was no shock to be

FIG. 3

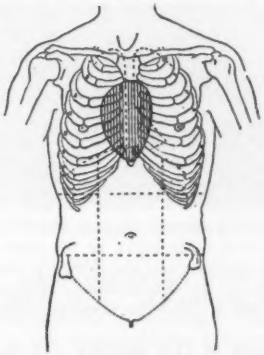


Chart showing area over which the diastolic murmur was heard on 10th January, 1905. (Dr. Cole.)

felt at the apex or base. At the apex the heart sounds were quite clear, both being well heard. The second was rather loud. Passing upward and inward in the fourth interspace, a soft, prolonged, blowing diastolic murmur became audible; this was heard over rather a wide area as shown in the diagram. Its maximum intensity was over the middle of the sternum, opposite the fourth interspace. The pulmonic second sound was not accentuated but was loud and well heard. The aortic second was also well heard but was not as loud as the pulmonic. There was no systolic murmur at the base.

FIG. 4

CASE III.—Radial pulse, 10th January, 1905.

11*th January.* Yesterday evening the patient began to complain of sharp abdominal pain which could not be definitely localized and was relieved by movements of the bowels. Colorless corpuscles, 15,400 to the cubic millimetre.

Cultures were again taken from the blood; 20 c.c. of blood were taken from the median basilic vein under antiseptic precautions and divided among six plates of agar, one plate of litmus milk and one flask of bouillon.

13*th January.* At the morning visit Dr. Osler made the following note: "The patient looks pale, the tongue is red and fissured; pulse is full, soft, dicrotic. The skin is clear; no petechiæ.

"*Heart.* Apex beat not visible; no marked pulsation in the vessels of the neck. There is a wavy impulse in the veins of the arm. In addition to the distinct pulsation there is a filling of the vein with expiration and the partial emptying with inspiration. There is no shock of either sound at apex or base; no forcible impulse; no especial impulse in the cardiac area. On auscultation, at the apex both sounds are heard. There is a soft murmur with the first sound. Toward the base and along the left sternal border there is a diastolic murmur which increases in intensity and is at its maximum in the second left space; it is well heard also at the second right costal cartilage and over the manubrium; well heard down the sternum. The pulse has become of a collapsing quality."

On the following day (14th January) Dr. Osler noted that the apex beat was faintly visible in the fifth left interspace inside the mammillary line; not palpable. The shock of neither sound was palpable. No thrill at the base. A wavy impulse in the neck but not of the character of aortic insufficiency. "Visible pulsation in the upper arm is quite marked. Radial pulse has become collapsing. At the apex both sounds distinct, somewhat increasing in intensity toward the sternum. No systolic heard at the apex. Sounds quite loud at the lower sternum. On approaching the third rib there is a diastolic murmur of great intensity, which, to-day, has definitely changed in quality. It is not only more intense but has a decidedly musical sound. It is heard well also at the aortic cartilage."

Results of cultures taken on 11*th January* On the plates containing equal quantities of agar and blood there was in twenty-four hours, at 37° C., a growth of faint grayish translucent colonies, deep in the medium; all flat and round—not oval. On blood agar, surface smears, there was a good, discrete growth of fine grayish translucent colonies. On hydrocele agar there was a growth of discrete, grayish, translucent colonies.

These colonies all consisted of a biscuit-shaped diplococcus without motility, staining readily and evenly and decolorizing by Gram's method.

After forty-eight hours there was no growth on the surface of the plates of blood and agar. The colonies already present were 1 to 2 mm. in diameter—no subsequent increase in size. On the blood agar surface smears the colonies in forty-eight hours were not over 2 mm. in diameter. On blood serum in forty-eight hours there were faint discrete colonies. On hydrocele agar, in forty-eight hours, the colonies were slightly larger—some fusing of the colonies. There was no growth on plain agar slant, on hydrocele fluid and bouillon or on litmus milk.

Diagnosis: *Gonococcus.*

On the following day (16th January) Dr. Cole noted that the area of cardiac dulness reached 2.5 cm. to the left of the midsternal line and 4 cm. to the right in the fourth interspace. The spleen was not to be felt.

17*th January. Blood Count.* Red blood corpuscles, 4,640,000 to the cubic millimetre; colorless corpuscles, 14,800 to the cubic millimetre; hæmoglobin, 82 per cent.

The patient steadily lost ground. On the 18th he complained of epigastric pain and had several attacks of nausea and vomiting. On this date the following note was made by Dr. Thayer: The patient looks rather pale and ill, the tongue is very red and beefy; pulse, regular, of large size, short duration, ill sustained, 25 to the quarter. There is moderate heaving in the precordial area. Apex impulse, not clearly defined. The cardiac dulness begins above at the upper border of the third rib and extends about 4 cm. to the right of the midsternal line and, over the fifth rib, about 8 cm. to the left. The greatest diagonal dulness from left to right measures a trifle over 13 cm. The sounds

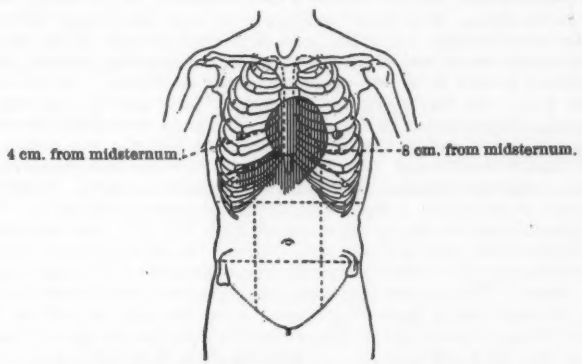
are best heard in the fifth interspace just inside the outermost dulness. At the apex the first sound is clear, the second followed by a soft, blowing diastolic murmur which is prolonged through the greater part of diastole and, in the fourth space at about the parasternal line, becomes louder and more accentuated in the latter part of diastole, taking on the features of a mitral presystolic murmur. Along the left border of the sternum the first sound is clear

FIG. 5

CASE III.—Radial pulse, 16th January, 1905.

the second, replaced by a well-marked diastolic murmur which is heard loudly in both aortic and pulmonic areas. The element of the second sound is louder in the pulmonic than in the aortic area. The murmur is well heard over the manubrium. In the carotids there is only a rather faint systolic tone; slight tone in the femorals. It is rather striking that the presystolic murmur is so well heard inside the parasternal line, as well as in the tricuspid area.

FIG. 6



CASE III.—Showing cardiac dulness on 18th January, 1905.

The nausea and vomiting continued. On the 20th Dr. Osler noted that the murmur had increased greatly in volume and intensity. No systolic murmur was audible.

On the 21st I saw the patient again, noting that the pulse was markedly dicrotic, ill-sustained, though not of a particularly large size nor of a characteristic aortic character. "At the apex but two sounds are heard; a long and rather rumbling murmur with a short sound of moderate intensity. The short sound is the first sound. The long rumbling murmur occupies, appar-

ently, all diastole. Passing inward, the diastolic murmur becomes greatly increased in intensity, loudest at the sternal end of the fourth left interspace, where the first sound is barely audible, the loud roaring murmur being almost the only sound. In the second left interspace the second sound is fairly sharp and the murmur is of less intensity. In the aortic area there is a to-and-fro murmur of moderate intensity. An element of the second sound is heard. It is interesting that, at the apex, the murmur, though feebler than near the sternum, runs directly into the first sound. Near the sternum it reaches its climax and comes to an end a little earlier. The right side is much enlarged, reaching, on percussion, as much as 6 cm. to the right of the midsternal line. The dulness on the left reaches about 9.2 cm. outward over the fifth rib. There is a slight femoral tone. The liver flatness reaches 7 cm. below the tip of the ensiform and 4 cm. below the costal margin in the mammillary line."

On 23d January, Dr. Osler noted a distinct, slight vibratory thrill, and on the following day Dr. Cole described a well marked thrill.

Blood Count. Red blood corpuscles, 4,108,000 to the cubic millimetre; colorless corpuscles, 13,200 to the cubic millimetre; hæmoglobin, 72 per cent.

The temperature was very irregular, intermittent and remittent. Febrile paroxysms frequently accompanied with chills, occurred sometimes as often as three times in twenty-four hours.

On the 27th Dr. Thayer noted that the pulse was 30 to the quarter at the time of the visit; the right side of the heart extended on percussion about 5.8 cm. from the median line; on the left only about to the mammillary line or 10 cm. from the median line. The first sound was heard best in the fifth space a little inside the point of outermost dulness; it was not loud but was rather sharp; the second sound was immediately followed by a diastolic murmur which ran all the way up to the first sound, having, toward the end of diastole, something of a rumbling quality. In the fourth left interspace the murmur was louder and more like a mitral diastolic. It became still louder as one passed inward and was loudest a little outside of the left sternal border in the fourth space. The heart's action was so rapid that it was difficult to place the sound exactly, but at the point of greatest intensity of the diastolic murmur at the sternal and of the fourth space, the rumbling, loudest part of the murmur seemed to be late rather than early in diastole. At this point the first sound was very slight and the second barely audible, marking the beginning of the murmur. In the pulmonic area, in the second left interspace, there was a soft to-and-fro murmur in association with the two sounds, the second, sharply accentuated. The same is true in the aortic area, the murmurs, however, being rather louder. The second aortic sound is sharp. There is no sharp tone in the aorta; a tone in the crurals of moderate intensity. "The striking features of this case are an extremely large right side with auscultatory signs which would point to a pure aortic lesion. On the other hand, the first sound at the apex has a rather short and abrupt character which might suggest mitral disease. The position of greatest intensity of the late diastolic murmur in the tricuspid area is again remarkable. Over the right side of the chest and the dull area to the right of the sternum the heart sounds are well heard, each followed by a soft murmur. At the end of the third left interspace the diastolic murmur is limited to early diastole, while over the whole of the rest of the left precordial area there is a late diastolic accentuation. The pulse is collapsing and of aortic quality."

The patient steadily lost ground; there was intermittent fever with frequent chills. Examination of the fresh blood on January 29th showed that the red blood corpuscles were of normal size; a little pale; no malarial parasites. There was a moderate leucocytosis with a moderate increase in polymorphonuclears.

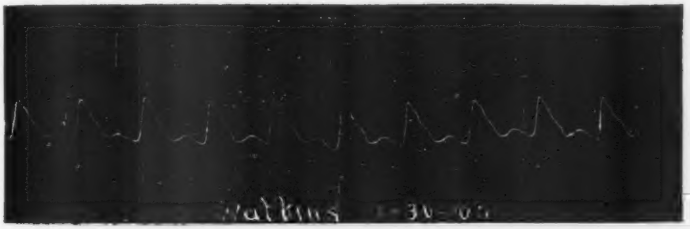
30th January. Dr. Osler observed on this date, that "the most interesting change that has occurred is the rough diastolic over the body of the heart. Over the aortic area and along the left border of the sternum the murmur is

soft, the maximum intensity being in the tricuspid area. There is a good deal of pulsation in the neck.

31*st January*. *Blood Count*. Red blood corpuscles, 3,164,000 to the cubic millimetre; colorless corpuscles, 25,000 to the cubic millimetre; hæmoglobin (Gowers), 60 per cent.

The patient steadily lost ground. On 2d February Dr. Cole noted that there had been no special change in the heart sounds excepting that the

FIG. 7



CASE III.—Pulse tracing, 30th January, 1905.

rumbling murmur in the fourth and fifth interspaces inside the parasternal line had become more marked and more typically presystolic in time, of entirely different quality from the diastolic murmur heard over the sternum and along the left sternal margin.

On 3d February Dr. Thayer noted that the patient looked puffy and doughy. There were a number of perfectly fresh petechial spots on the trunk and

FIG. 8

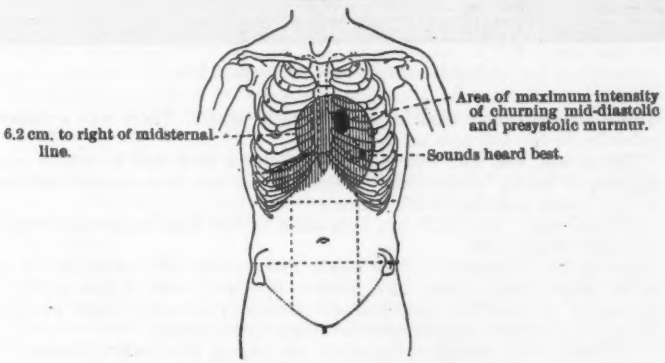
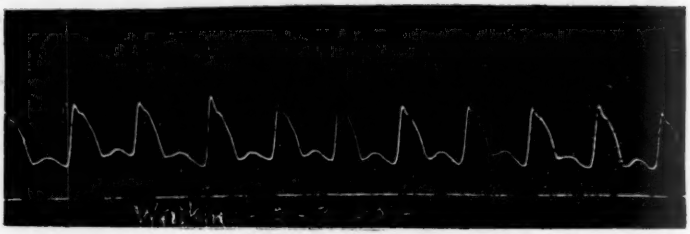

Area of maximum intensity of churning mid-diastolic and presystolic murmur.
6.2 cm. to right of midsternal line.
Sounds heard best.

Chart made by Dr. Thayer on 3d February, 1905.

extremities. The pulse was 29 to the quarter, soft and of rather good size; of short duration; collapsing quality marked on raising the wrist. The pulsation of the carotids was fairly well marked. The apex impulse was not sharply localizable. Slight impulse just behind the left nipple. There was epigastric pulsation. On percussion the dulness extended 6.2 cm. to the right of the median line in the fourth space, and 5 cm. in the third. In the apex region the sounds were best heard in the fifth space, about 7.5 cm. out. There was

a gallop rhythm dependent upon a reduplication of the first sound or a very slight presystolic murmur, it was difficult to say which, as well as a very slight systolic murmur. The sounds were very soft and masked by loud stertorous breathing. The second sound which was audible was followed by a very slight diastolic murmur. Passing inward a murmur became heard in diastole leading up to the first sound and beginning, apparently, immediately after the second sound. In the fourth space, inside the parasternal line, this became suddenly very loud and near the ear, of an echoing, vibratory quality and was almost the only sound heard. Listening very carefully a slight soft whiff of a different character might be heard at the beginning of diastole. Immediately above this the rumbling murmur disappeared and the first and second sounds were heard, followed by a soft diastolic murmur limited to the early part of diastole and of ordinary aortic quality. The second sound in the second left interspace was sharp and associated with this same diastolic murmur. In the second right interspace was heard a short systolic souffle and a fairly sharp second sound followed by a diastolic murmur. The churning mid-diastolic and presystolic murmur was heard with maximum intensity in the limited area marked on the chart in the third and fourth left interspaces. To the right of the sternum, in the dull area, the heart sounds were well heard—with a well marked to-and-fro systolic and diastolic murmur.

FIG. 9



CASE III.—Radial pulse, 3d February, 1905.

These were especially loud in the third right space. There was a distinct pulsation in the dull area on the right.

The patient, who had been failing rapidly for a week and constantly complaining of feeling "awful sick," became weaker and more restless and died at 7.10 on the morning of 4th February.

The necropsy (No. 2463) was performed by Dr. MacCallum three and a half hours after death.

Anatomical Diagnosis: Acute aortic endocarditis with ulceration of the valves; acute splenic tumor with anæmic infarction; acute diffuse nephritis; myocardial degeneration; atelectasis and œdema of the lungs; slight cirrhosis of the liver; chronic adhesive pleuritis; diphtheritic cystitis.

"*Thorax.* The pleural cavity of the left side is free from adhesions but contains about 600 c.c. of turbid fluid, and the right pleural cavity is almost obliterated by thin veil-like adhesions. The pericardial cavity contains a little thick fluid. The surfaces are smooth and glistening. The heart is somewhat enlarged. The right ventricle is extremely flabby, and as the heart lies in the pericardium, a solid opaque mass can be felt through the walls of the right ventricle, projecting so as to be easily seen. It is apparently attached to the wall of the ventricle which cannot be lifted away from it.

"*Heart.* The heart is smooth externally and there is a small hæmorrhage over the anterior portion of the back of the ventricle. There is a similar small

hæmorrhage on the posterior surface near the apex. The tricuspid valve, viewed from the auricle, shows no abnormality. The mass described above proves to be a papillary muscle. The tricuspid and pulmonic valves are quite normal. The mitral orifice measures about 3 cm. in transverse diameter. Viewed from the auricle no definite vegetations are seen. The valve is thickened throughout its whole edge except for a small portion of the posterior valve. The posterior valve is much shortened. The thickening is most marked in the anterior leaflet, where it extends for a width of perhaps 7 mm. back from the edge. The chordæ tendineæ are practically normal. The aortic valves are covered on the ventricular side with a vegetation which involves the right and left coronary segments. The vegetations are large and rounded, measuring perhaps 1.5 cm. in diameter. They spring from the central portion of each of the valves and bind the two valves together so that the left coronary segment is much distorted and pulled over toward the right. A probe can be easily passed through the valves where the vegetations lie upon them, and thus into the ventricle.

"When the fresh clot is removed the vegetations are found to be very rough and irregular and covered with ridges. The holes in the valves are at least 6 mm. across. The valves are obviously eroded when viewed from the sinus of Valsalva, the eroded portion being roughened and covered with red clot. The left coronary artery shows some yellow plaques of sclerosis, but it is patent and wide. The right coronary artery is much smoother and shows no extensive plaques of sclerosis. The back of the mitral valve shows some yellow patches. The aorta is fairly smooth, there being some sclerosis and a thickening just above the valves. The myocardium of the right ventricle is excessively flabby. The wall of the ventricle measures 3 mm. in thickness. The myocardium of the left ventricle measures about 15 mm. in thickness; it is rather opaque and dull gray in appearance.

"The *spleen* measures 16 x 14 x 6 cm. and weighs 550 grams; it is very much enlarged. Its capsule is tense and dark grayish-red in color. The upper surface of the spleen shows two patches of dense, firm tissue which are slightly elevated above the surrounding surface and are grayish yellow in color with a margin of blackish red. Both of these areas are covered with a fibrinous exudate. On section they extend into the substance of the spleen and are opaque, grayish yellow in color. The larger is distinctly softened in the centre. The spleen is very much swollen and on section is rather grayish looking and opaque. The Malpighian bodies stand out sharply as opaque gray dots in an excessively hypertrophied splenic pulp.

"The *bladder* is distended with urine. In the posterior wall just below the fundus there are numerous red areas somewhat raised, dull on the surface, pale in appearance and surrounded by a margin of deep red spots. The mucosa of the bladder elsewhere is fairly normal, somewhat dull and granular at the trigone.

"The *prostate* is not enlarged but the prostatic veins contain, here and there, thrombi. The seminal vesicles are normal in appearance and contain a clear fluid. The testicles and epididymes are apparently normal."

Microscopically, the urethral mucosa was slightly infiltrated with leucocytes, while the mucosa of the bladder showed a typical diphtheritic inflammation.

Microscopical examination of the vegetations on the aortic valve showed a large number of diplococci of distinct biscuit-shape, most of which were intracellular. Some cells were packed full of cocci. The organisms decolorized by Gram's method.

Cultures on ordinary media from the heart and kidney were sterile. Those from the spleen showed one small colony of *streptococcus pyogenes;* from the abdominal cavity, one colony of *streptococcus pyogenes* and one of *micrococcus albus*. From the bladder which had been opened and somewhat exposed, a small bit of tissue was excised after singeing the surface, and put into the culture

medium. Colonies of the *pseudodiphtheria bacillus* and *bacillus alcaligines* were obtained. Ascitic agar tubes were made from the vegetations on the aortic valve. On these there developed in pure culture, small, white colonies of biscuit-shaped diplococci arranged in tetrads and pairs which decolorized by Gram. No growth in milk. Diagnosis: *Micrococcus gonorrhœæ.*

At the same time Dr. Boggs made cultures from the heart's blood and from the vegetations on the aortic valve—smears on hydrocele agar. Those from the heart's blood were negative; those from the vegetations showed a pure culture of *micrococcus gonorrhœæ.*

This case was particularly interesting from the point of view of the physical signs. The remarkable presystolic accentuation of the diastolic murmur, the intensity of the presystolic rumble, the area in which it was heard, and the great extent of the dulness to the right would seem to have quite justified the suspicions which we had *intra vitam* of the existence of a combined aortic and tricuspid lesion. From the anatomical lesions, however, it would seem that this murmur must have been due to the disturbance caused by the regurgitation of a large mass of blood directly upon the anterior mitral curtain at the time of entry of the blood from the left auricle.

* * *

B. Four Cases of Ulcerative Endocarditis following Gonorrhœa.

CASE IV.—*Male, aged twenty-one years; gonorrhœa; arthritis; ulcerative endocarditis of the mitral valve; embolism of the anterior tibial artery; death; no necropsy.*

X., aged twenty-one years, a commercial traveler, was seen with Dr. Slemons, of Salisbury, Maryland, on March 18, 1899.

His *family history* was good. He had always been a healthy boy; had never had rheumatism or serious illnesses of any nature.

Present Illness. In the latter part of December the patient contracted gonorrhœa. Early in January, he began to have chills at irregular intervals followed by fever and sweating. After the first several chills the discharge disappeared. He kept about at work despite the fact that he felt ill. In February the right knee and ankle became somewhat swollen and painful and he had to give up work for several days. In a severe storm early in February he was subjected to great exposure, and on the 20th the chills and fever, which had continued, had so far exhausted him that he was obliged to give up work and go home. Since this time he had been in bed suffering from fever which, at times, had been more or less continuous, at other times intermittent and associated with excessively severe rigors. The duration of fever, during these paroxysms was often surprisingly short, the patient having sometimes as many as three attacks in twenty-four hours, the temperature falling with great rapidity in association with profuse, drenching sweats. He had become feeble and anæmic. The febrile paroxysms were often accompanied by vomiting. Several days before my visit, during a paroxysm, the patient suddenly felt a numb, tingling sensation in the right leg. Since then the leg below the knee had felt as if it were asleep; it was cold and he had been unable to move it as freely as the other, while the foot and calf had been somewhat tender.

At the time of examination the patient was in bed, on his back; well formed man; somewhat emaciated. The face was pale and sallow; the lips and

mucous membranes, pale. There was marked pulsation of the vessels of the neck. The pupils were widely dilated; the respirations rapid and rather shallow. The patient looked very ill and was so weak that he was unable to tell a connected story of his illness, the effort to speak being very considerable. The tongue showed a black coat (peptomangan). The pulse was regular, of low tension, 33 to the quarter. The *lungs* were clear throughout.

Heart. Well marked pulsation in the fifth space just inside the nipple. On palpation there was a marked systolic thrill in the pulsating area. Dulness began at the second space above and did not reach the right parasternal line. No marked evidence of right-sided enlargement. On auscultation there was a very loud, blowing, systolic murmur which almost replaced the first sound at the apex and was heard throughout the axilla and back. The murmur diminished markedly as one passed inward and upward, but a soft systolic murmur, probably the mitral murmur transmitted, was heard all over the cardiac area. The second pulmonic sound was accentuated; the second aortic feeble but clear. In the tricuspid area the first sound was well heard in association with the murmur. The *spleen* was not palpable, though the area of dulness was markedly increased. *Liver* not remarkable. No ascites. In a rather poor light no petechiæ were visible.

The *right leg* below the knee was pale and distinctly cold as compared with the left. The posterior tibial artery was not palpable on either side. The pulsation of the anterior tibial was felt on the left but not on the right.

Urine (only one ounce obtained), high color; trace of albumin; sediment, on centrifugalization: Decolorized red blood corpuscles; leucocytes in considerable number; hyaline and finely granular casts with red blood cells and fatty degenerated epithelial cells adherent. Occasional free, fatty degenerated epithelial cells.

Blood. Fresh specimen. The corpuscles are pale; difference in size, more than normal; slight poikilocytosis; great increase in the number of leucocytes.

A differential count of a thousand leucocytes stained by Ehrlich's triple stain, showed small mononuclear leucocytes, 4 per cent.; large mononuclear and transitional forms, 5.6 per cent.; polymorphonuclear (neutrophiles and two or three mast cells), 90.1 per cent.; eosinophiles, 0.3 per cent.

No nucleated red blood corpuscles were seen. No cultures from the blood could be taken. The patient steadily lost ground and died within a week after the visit. There was no necropsy.

In this instance the direct association between the acute gonorrhœa and the development of the ulcerative endocarditis would make it in every way probable that the case was one of true gonorrhœal septicæmia.

CASE V.—*Male, aged thirty-five years; gonorrhœa; ulcerative endocarditis of the mitral valve; death; an unidentified bacillus obtained in cultures from the valves.*

X., male, aged thirty-two years, a lawyer, was seen in consultation with Dr. H. B. Deale, of Washington, on 2d June, 1899.

The patient had had the usual diseases of childhood. Four years ago, severe attack of typhoid fever lasting five weeks. Has been under treatment for several years for some throat affection.

On 1st April, after a hard winter, he began to feel very tired and thinks that he had an irregular fever coming on particularly in the afternoon, and lasting from three to five hours, followed by profuse sweating. The fever was occasionally preceded by a slight chill lasting from fifteen to forty-five minutes. There have been frequent headaches; loss of appetite; moderate diarrhœa. On further inquiry it was found that the patient had had gonorrhœa at about the time of the onset of his symptoms.

The patient was first seen by Dr. Deale on 13th May, 1899. The temperature was 103°, pulse 88. There was nothing abnormal about the heart sounds or quality of the pulse. The blood showed no malarial parasites. The fever ranged from 99° to 103°, the pulse from 76 to 104 during the first week.

On 18th May a loud systolic murmur was heard at the apex, transmitted to the axilla. About 27th May the temperature became higher and the pulse much more rapid with periods of marked irregularity. Widal tests on 20th and 24th May were negative.

2d June. The patient was an emaciated, anæmic-looking man, somewhat cyanotic; respirations rapid and rather shallow; vessels of the neck pulsating markedly. Pulse 34 to the quarter, partly from excitement, of rather low tension. *Heart.* The apex impulse was palpable about in the mammillary line. There was a loud systolic murmur which almost replaced the first sound, and was heard well through the axilla and back. The murmur was barely audible at the base; the second pulmonic sound sharply accentuated. At the tricuspid area and over the greater part of the right ventricle the second sound was somewhat reduplicated, having a sticky character; slight suggestion of a diastolic murmur. The second aortic was feeble but clear. The *lungs* were clear on percussion. On auscultation, fine, sticky râles were heard at both apices and bases, and, to a certain extent, along the anterior borders of the lungs, while the respiration over the middle of the right back was rather feeble. The *spleen* was not palpable, although the area of dulness appeared to be increased. The diagnosis was ulcerative endocarditis of the mitral valve, probably gonorrhœal.

The patient died on 3d June.

The necropsy by Dr. Carroll showed that the heart was much enlarged; the left ventricle markedly hypertrophied. The walls of both ventricles were thickened. The aortic valves were competent with the water test and quite normal. "Both flaps of the mitral valve support large, irregularly shaped, pendulous masses of new formation which project from one-fourth inch at the margin, to about three-fourths inch at the centre, into the cavity of the ventricle. The leaflets of the tricuspid valve showed a slight nodular thickening along their margins. Cultures from the heart's blood, vegetations and from the liver on plain agar, human serum agar, Loeffler's blood serum mixture, blood agar mixture and bouillon revealed small, slowly developing, circumscribed colonies of small, slender bacilli often in pairs. These failed to grow on any transplantations. They were decolorized when treated by Gram's method. One of the Loeffler tubes from the heart's blood and one blood agar mixture showed these organisms in pure culture. The same organism was found in cultures from the vegetations in connection with a staphylococcus."

The close association of the onset of symptoms in this case with an attack of gonorrhœa justifies the suspicion that the urethritis may have served as the portal of entry to the infection, although evidence of its primary gonorrhœal nature is wanting.

CASE VI.—*Man, aged thirty-one years; gonorrhœa; prostatitis; arthritis; ulcerative endocarditis (mitral insufficiency); death; no necropsy.*

H. G., male, aged thirty-one years; clerk. Family history good. Had always been a strong, robust man, rather given to athletics. Had never had any serious illness or previous venereal disease. Consulted Dr. S. Likes on 7th July, 1902, with a gonorrhœa of eight weeks' duration, which had been treated by internal and local medication. Examination showed no discharge from the meatus. The urine was very cloudy by the test of the three glasses. The prostate was uniformly enlarged and the secretion following massage was very purulent and contained gonococci. The patient was put under

treatment—daily massage of the gland, followed by mild permanganate irrigations. He began to improve until the prostatic secretion was almost normal and the urine clear.

On 24th July he had a chill followed by pain and swelling in both knee-joints. The chills recurred at irregular intervals, though the rheumatic symptoms soon began to abate, disappearing completely within a week. No local lesion could be found to account for the symptoms.

On 12th August I saw the patient in consultation with Dr. Likes. He was a healthy looking man, though the complexion was a little dull and sallow. The lung and heart sounds were clear. The spleen was palpable. The urine was free from albumin.

The chills and irregular fever continued and the patient was seen again two days later. At that time a soft systolic murmur was audible all over the cardiac area, rather harsher, perhaps, in the aortic area. The two sounds were of normal relative intensity.

Ten days later the patient was seen again. Irregular fever and chills had continued, although the patient felt quite well between attacks. The patient began to look somewhat anæmic and was clearly losing weight. The pulse was large and soft. The systolic murmur was audible, loudest at the apex, clearly heard as far outward as the anterior axillary line. The second pulmonic was accentuated; the apex impulse was 11.5 cm. from the median line. A specimen of *urine* was of normal color, acid, 1015, showing a slight trace of albumin.

From this time on the patient steadily lost ground. I saw him again on 10th October, at which time he had become more emaciated and anæmic. A systolic murmur similar to that before described was still audible. From this time the patient's condition grew gradually worse, chills recurring once or twice in twenty-four hours, although at one time there was a period of four days of normal temperature. From time to time Dr. Likes states there were attacks of violent coughing lasting about twenty-five minutes—about six of these attacks in all. There was marked and progressive anæmia with enlargement of the spleen and local tenderness. At no time was there any pain over the cardiac area. The patient gradually lost ground and died on November 9th, one hundred days after the onset of the condition. Dr. Likes states that the cardiac murmur became louder in the latter days of the illness. No necropsy was allowed.

In this instance no cultures were taken from the blood, but the onset and course of the case were strongly suggestive of a true gonorrhœal endocarditis.

CASE VII.—*Man, aged twenty-one years; urethritis; epididymitis; intermittent fever; ulcerative aortic and pulmonic (?) endocarditis; arthritis; cerebral embolism; streptococcus pyogenes obtained from the peripheral circulation; death; no necropsy.*

Y., aged twenty-one years, seen in consultation with Dr. Y. on 27th October, 1903. Family history unimportant. Has been a healthy boy. Has never had rheumatism. In January the patient had an attack of urethritis followed later by an epididymitis from which, in the course of several months, he apparently entirely recovered. He has not, however, been well since this time. During the summer, especially in the latter part, he has had slight recurrent febrile attacks.

On 6th September he came home from Chicago and was put to bed by his father, who is a physician. The temperature ranged from 97° to 99° in the morning to 102° to 103° at night. There was considerable sweating. The heart was enlarged and tumultuous in its action; pulse, 90 to 110; respiration, 25 to 30. The appetite was good. He complained at times of soreness in the knees and muscles of the legs.

The patient, seen in the evening, was in bed; skin hot and moist; large frame, rather sparely nourished; somewhat pale and of a slightly yellowish complexion. Pulse, large and collapsing, suggestive of aortic insufficiency.

Heart. Well-marked heaving in the precordial area. The apex was in the fifth space about 13.5 cm. from the median line, outside the mammillary line. Dulness to the right, not especially increased. The impulse was heaving. On auscultation the first sound was dull and indistinct; the second, replaced by a slight diastolic murmur which became more marked along the left border of the sternum and was heard most clearly in the third and fourth left spaces close to the sternum; it was also heard in the aortic area. The first sound in the aortic and pulmonic areas was indistinct. Passing out over the lungs on either side a well-marked systolic murmur became audible, which was not distinctly heard anywhere in the precordial area. This was heard everywhere in the chest, front and back, in the erect and recumbent postures. The lungs were clear throughout. The spleen was just palpable. One or two petechial spots were to be made out on the chest and arms.

Urine. Amber color; acid; specific gravity, 1025; no sugar; albumin possibly a slight trace; sediment, very slight cloud; microscopically, a few hyaline casts; a few pus and epithelial cells. Centrifugalized specimen, stained for diplococci; none found.

A *blood* culture was taken from the right arm, about 6 to 7 c.c. of blood being drawn from the median basilic vein and immediately plated with melted agar. From this plate numerous large and small colonies grew, which were found to contain a large spore-bearing bacillus, doubtless a contamination. At the time of this examination I made the following additional note: "The remarkable feature of this case is the systolic murmur heard over the lungs. The only instance in which I have observed a somewhat similar occurrence was that of Mr. B. at the hospital, who had a systolic murmur well heard over the left lung and not in the precordial area, which was supposed to be due to an aneurysm. At autopsy it was found that it was dependent upon a stenosis of the main branch of the pulmonary artery at the root of the lung owing to an extensive gumma. May the murmur in this case be due to vegetations situated in the pulmonary artery above the valves?"

A diagnosis was made of ulcerative endocarditis of the aortic valves, possibly of the pulmonic as well.

On 1st November, a letter was received from his father stating that on the morning of 24th November, "The patient, without any warning, was suddenly seized by aphasia." This, as a paraphasia, continues without improvement. Coincident with it he has at times some pain over the region of the third frontal convolution. Muscular soreness has been at times very prominent, no delirium or mental aberration. At times some mental depression and drowsiness. A large number of petechiæ have appeared over the body very small in size.

On 2d November, Dr. Howard saw the patient who was pale; cachectic; emaciated; mind clear; marked sensory aphasia; no evidence of pain in any of the muscles. The right knee on the inner aspect is slightly red, hot, tender and puffy as also are the right first and second metacarpo-phalangeal joints.

Heart. Point of maximum impulse in the sixth space outside the mammillary line. Impulse strong and heaving; no thrill. Relative cardiac dulness, increased to the left; almost 4 cm. outside the mammillary line. At this point there is a loud blowing systolic murmur transmitted to the axilla and over the body of the heart. The second is faintly audible. At the base there is a soft diastolic murmur, of maximum intensity in the second right interspace. No accentuation of the second. Pulse, regular, definitely collapsing. *Cultures* from the blood, taken in the same method as before, revealed a pure culture of *streptococcus pyogenes.*

The patient steadily lost ground and died within the course of a week.

In this instance again the relation of the ulcerative endocarditis to the gonorrhœa, while suspected, cannot be said to be positively proven. The history of its development following the complications of a severe urethritis is, however, a noteworthy circumstance, justifying the suspicion that, whether or not the process were primarily gonorrhœal, the urethritis may have been the portal of entry for the infection.

REPORT OF A CASE OF PNEUMOCOCCUS SEPSIS.

By John S. Thacher, M.D.,
of New York City.

This case is presented as an illustration of the terrible ravages which the pneumococcus may produce throughout the human body in a very short space of time.

The patient, a man, aged forty-five years, of excessive alcoholic habits, though a gentleman, was sick only nineteen days. During the first twelve days he went through an attack of acute lobar pneumonia of severe type. There was then a distinct improvement, not by crisis, however, but gradual and not complete. The signs of pneumonia shrank to a very small area.

Five days before his death he showed the first signs of fluid at the base of the same lung, which later proved to be purulent.

Two days before his death the first signs of heart involvement appeared, diastolic murmur and a large, soft pulse with a thrill, followed later by others and loud murmurs, the heart at autopsy showing marked ulcerative endocarditis.

The same day, two days before death, the first signs of meningitis appeared—severe headache and congested eye—but this complication was not suspected until the following day, the day before death, when the neck was stiff. The autopsy showed a very intense meningitis.

The fluid from the chest and from the spinal canal, as well as the blood taken during life, showed an abundant pure invasion of pneumococci.

Taking the illness now in detail, beginning with the pneumonia, we find that on the first day he simply felt a little miserable. On the second day he had a chill, was prostrated, ached throughout the body and took to his bed. He had on that day a little cough, with some expectoration, which seems to have been mucopurulent. On the fifth day of his illness he became delirious, with great mental depression. It was not until the sixth day that he felt pain in his chest, which was quite sharp on deep inspiration. On this day, the sixth of his illness, he came into the hospital very sick. He was

irrational, very tremulous, much prostrated, with considerable dyspnœa and cyanosis. The pulse was, however, strong, regular, of good size and soft, with a rate on that day between 90 and 100.

The examination of the lungs discovered typical signs of pneumonic consolidation of the right lower lobe, with numerous subcrepitant rales. There was also a faint bronchial character to the expiratory murmur over the posterior part of the upper lobe on the same side, and over the posterior part of the left upper lobe and the upper part of the left lower lobe, but this was very likely a transmitted quality from the consolidated right lower lobe, and no development of disease followed in these parts. The abdomen was distended, hard, and tympanitic. On the following day, the seventh of the illness, he is reported to have stopped breathing for one minute at 5 A.M., but he resumed that function without further interruption.

During the first four days in the hospital, that is, the sixth to ninth days of the illness, he was an extremely sick man, delirious, dyspnœic, cyanotic; the temperature ranging gradually higher, up to 104°, the pulse becoming more rapid, though only to about 120 and less strong.

The first sign of improvement was in the abdomen; on the last of these days, the ninth of the illness, it became softer. The following day, the tenth, there was distinct improvement; he became quiet and rational; the temperature came down to 101.5°; the pulse to 96. The next day the signs of consolidation had diminished in area, and on the day after that, the twelfth of the illness, had shrunken to a small area just inside the angle of the scapula. The pulse at this time had improved, its rate coming down on the thirteenth day to 84, while the temperature, with some irregularities, came down to 101.5°.

On the fourteenth day, however, signs of fluid appeared in the right chest, dulness at the base, and faint bronchial breathing over the whole of that left lower lobe. The temperature also began to rise again, and the signs of fluid advanced. Two days later the dulness was noted as higher; there was diminished bronchial voice and breathing, and diminished fremitus at the base without any rales. The heart apex also began to move toward the left, being now four and three-quarter inches to the left of the middle line, instead of four and one-quarter inches, where it was located on the day of his admission. The next day the dulness extended to the angle of the scapula, and the voice at the middle scapular region had a nasal quality. There was very faint bronchial breathing over the back of the left upper lobe, but this was believed to be transmitted from the right side. The apex beat was now five and one-quarter inches to the left of the middle line. The right pleura was aspirated on this day, and 300 c.c. of turbid, greenish-yellow fluid obtained. This fluid, after standing, showed 50 per cent. of

puruloid sediment and contained pneumococci and no other kind of bacteria. The pneumococci were identified by their characteristic microscopic appearances, including the capsules, and by the coagulation of Hiss' inulin medium.

The apex beat was found on the next day to have returned to a point four and one-quarter inches to the left of the middle line—that is, to the position which it occupied on the day of admission—one inch within its position before aspiration. The lung became resonant on percussion again down to the base, the respiratory murmur, however, remaining faintly bronchial.

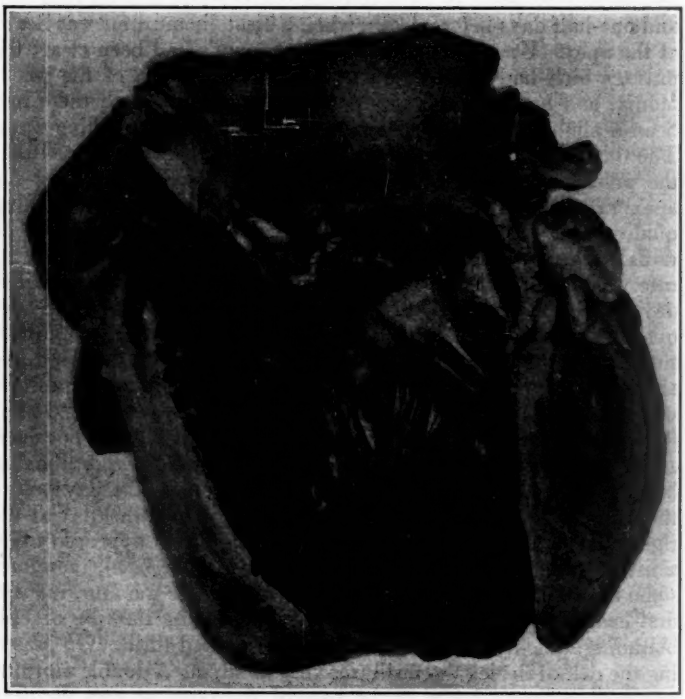
The first evidence of cardiac complication was noticed only two and one-half days before death, when a diastolic murmur was heard at the apex. Up to that time the heart sounds had been clear; the pulse, which had quieted down with the resolution of the pneumonia, had not until this day passed 100 again, though there had been a slight increase in the rate during the two preceding days. The day before the murmur was first heard it had been noted that the pulse was good. This murmur, when first heard, was at the apex rather late in diastole, was very faint and of a rumbling quality, and was put down as an early presystolic murmur. The radial pulse became softer and had an appreciable thrill, but its rate was only 102. The apex beat on this day, as on the previous day, had been discovered to be moving outward from four and one-quarter inches to the left of the middle line, where it was on admission, to four and three-quarter inches on the previous day, and five and one-quarter inches on this day. These changes in position were probably due to empyema, for the apex impulse returned after the aspiration to its original position. The right border, however, at this time seemed to have moved a little toward the right, having been noted on admission as normal in position on the day before the murmur appeared as at the middle line, and on this day at the right edge of the sternum. On the following day it appeared to be a little further to the right. There seems to have been, therefore, some enlargement by this time. On the day after the murmur was first heard the pulse became more rapid, reaching the rate of 114, bounding, quickly collapsing, and with a marked thrill. On grasping the calf of the leg the thrill was very striking. A double murmur was now distinctly heard at the base of the heart on this the next to the last day. The next day, the patient's last, this double murmur could also be heard at the apex, and it was thought that a presystolic murmur was also heard.

The first evidence of meningitis appeared two and one-half days before his death; that is, at the same time with the first evidence of endocarditis. On this day there was severe headache and congestion of the conjunctivæ of the left eye. An anxious expression, too, came into the patient's face. The next day the patient was in a semi-coma, being hard to arouse; the neck was stiff; there were movements of

the left leg seemingly automatic; Kernig's sign was not present. On the next day, the day of death, the neck became more rigid, the Kernig sign was present; a lumbar puncture withdrew three ounces of purulent fluid, which contained an abundance of pneumococci, presenting the same characteristics as noted in the cocci of the pleural fluid.

On the same day, before the patient's death, bouillon tubes were inoculated with the patient's blood, and characteristic growth of

FIG. 1



Left ventricle, showing vegetations on the aortic valve. Hairs pass through a small and a large perforation.

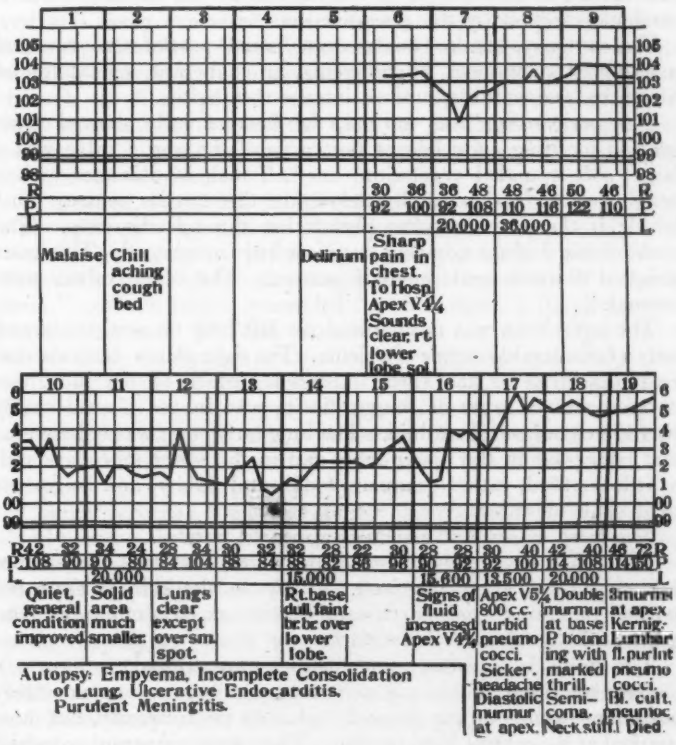
pneumococci developed in the bouillon, and, after transplantation, on blood serum and in Hiss' inulin medium.

During these last three days the general condition of the patient naturally became worse gradually and progressively, the temperature was most of the time between 105° and 106°, the pulse ran up to 150, and the respirations at one time during the last day were 72.

The leukocytes during his stay in the hospital were counted seven times and ranged between 13,500 and 36,000, the highest number being on the eighth day of his illness, the lowest two and one-half days before death, but on the day before death they rose again to 20,000.

The urine on the seventh day had a specific gravity of 1029, and contained 0.5 per cent. of albumin by bulk and some hyaline,

FIG. 2


Malaise
Chill aching cough bed
Delirium
Sharp pain in chest. To Hosp. Apex V 4/4 Sounds clear, rt. lower lobe sol
Quiet, general condition improved
Solid area much smaller.
Lungs clear except over sm. spot.
Rt. base dull, faint br. br. over lower lobe.
Signs of fluid increased Apex V 4/4
Apex V 5/4 800 c.c. turbid pneumococci. Sicker. headache Diastolic murmur at apex.
Double murmur at base P. bound ing with marked thrill. Semi-coma. Neck stiff.
Murmur at apex Kernig. Lumbar fl. purlnt pneumococci. Bl. cult. pneumoc
Died
Autopsy: Empyema, Incomplete Consolidation of Lung, Ulcerative Endocarditis, Purulent Meningitis.

Temperature chart with synopsis of symptoms.

granular, and epithelial casts. On the thirteenth day the specific gravity was 1016; no albumin or casts were found. On the sixteenth day the specific gravity was 1015; there was a very faint trace of albumin, but no casts.

Before the onset of this illness the patient is said to have been in good health, though often indulging too freely in alcohol. The

pneumonia, therefore, seems to have come upon him out of a fairly clear sky, and to have run a course not varying greatly from the ordinary except as modified by his alcoholic history, and to have resolved, though slowly and incompletely. When the pneumonia had nearly resolved an empyema developed, and immediately after this a general infection by pneumococci, which attacked the lining of the heart and the membranes of the brain and spinal cord so viciously that within a very few days extreme lesions were produced of ulcerative endocarditis and meningitis, the chief interest of the case arising from the rapidity and the intensity of the effects of the virulent infection by the pneumococci.

The autopsy, made twenty-seven hours postmorten, revealed a resolving pneumonia, an empyema, an exuberant vegetative and ulcerative endocarditis, and an intense meningitis.

The pericardium was normal, the heart muscle showed some grayish mottling. On one of the cusps of the aortic valve was a large, soft, rounded vegetation, one-half inch in diameter with a rough surface and a small perforation through its centre. Just beside it there was another perforation through the cusp. The under surface of the adjacent cusp is a little roughened. The heart weighed thirteen and one-half ounces. The other valves were normal.

The left pleura was normal and the left lung on section showed only a considerable degree of œdema. The right pleura, both visceral and parietal, over the entire posterior surface of the lung, was covered with a layer of shaggy fibrin, and in the pleural cavity was 175 c.c. of purulent fluid. The lung on section was œdematous and congested in the upper lobe, and in the lower lobe dark red, a little mottled, rather granular, tough and flabby, and fragments of it sank in water.

The liver, spleen and pancreas appeared normal.

The left kidney showed one little white infarct at the surface and running in about one inch. Otherwise the kidneys appeared normal excepting some grayness of the cortex on section and congestion of the veins of the surface.

The stomach, intestines, and bladder were normal.

Over the brain and the pia and arachnoid there was an extremely abundant exudate, quite general, including the convexity, but most marked at the central focal portion. There were numerous petechial hemorrhages in the walls of the fourth and lateral ventricles and a few drops of pus in the lateral ventricle.

Ten blood serum tubes were inoculated from the blood of the right and left ventricles and from the pericardium. On one of these inoculated from the left ventricle a growth appeared of pneumococci giving the characteristic reaction with Hiss' inulin medium.

INTRAPLEURAL LIPOMA; ACUTE PERICARDITIS; PERICARDIAL EXPLORATION.

By R. H. Fitz, M.D.,
of Boston.

It is well known that in certain individuals, especially the obese, a large quantity of fat tissue may be found beneath the pleura, and it is easily conceived that a lipoma might be developed from this subpleural fat. Cruveilhier,[1] indeed, mentions the occurrence of fatty tumors of the diaphragm which have forced their way through the fibres of this muscle from the subperitoneal fat, at times accumulated behind the ensiform cartilage. The attached peritoneum is stated to form a true hernial pouch ready to receive the abdominal viscera.

A rounded, lobulated, subpleural lipoma somewhat larger than a plover's egg was found by Clark[2] in the right pleural cavity of a woman, aged sixty-five years. Its "base was spread out between the muscle fibres of the diaphragm like the mycelium of a mushroom," and the tumor resembled "at first sight, a diaphragmatic omental hernia." He calls attention to a normal "outgrowth of fat from the diaphragm situated on the right side anteriorly close to the reflection of the pericardial pleura on to the diaphragm. This may, in cases of general obesity, become occasionally so increased in amount as almost to assume the appearance of a fatty tumor, but does not, of course, constitute a true tumor, since it is but part of a general physiological process."

Conner[3] records a somewhat similar observation. He found in an elderly woman who died of cerebral hemorrhage, a sessile, fatty tumor measuring 4 x 3 cm. It was covered by pleura and projected from the diaphragm into the left pleural cavity a little to the left of the apex of the heart. At its base there appeared to be a deficiency in the diaphragm, which was offered as a possible explanation of the origin of the tumor.

The subpleural fat along the intercostal spaces is often abundant, and Rokitansky[4] states that, at times, it projects into the pleural cavity as notable, lobulated masses.

Lancereaux[5] knows of but one well-authenticated instance of a subpleural lipoma. Chiari[6] is stated to have observed in an elderly

[1] Traité d'anat. path. gén., 1856, tome iii. p. 310.
[2] Transactions of the Pathological Society of London, 1887, vol. xxxviii. p. 324.
[3] Proceedings of the New York Pathological Society, 1897-98, p. 43.
[4] Lehrb. d. path. Anat., 1861, Bd. iii. p. 89.
[5] Traité d'anat. path., 1879-81, tome ii. p. 147.
[6] Gussenbauer, Arch. f. klin. Chir., 1892, Bd. xliii., Festschr. 223.

female a pendulous lipoma of the size of a walnut projecting into the pleural cavity from the seventh left rib.

The subpleural lipoma of the costal wall may become so large as to be of practical importance, as illustrated by the experience of Czerny.[1] This surgeon removed a lipoma of the size and shape of a man's head from below the left shoulder-blade of a male patient, eighteen years old. The tumor was of rapid growth and was found at the operation to be continued into the thorax between the seventh and eighth ribs, which were separated by a gap of the size of a hen's egg. The patient died of septic pleurisy, and the subpleural cavity within the chest from which the innermost portion of the tumor was removed, contained several sinuses. The growth was considered to have proceeded from the endothoracic fascia beneath the pleura. Several years later a like experience was published by Plettner[2] as having been met by Volkmann in the practice of another surgeon. An elderly woman had a lipoma of the size of a child's head projecting from the left wall of the thorax. At the operation it was found to be prolonged into the chest by a pedicle between the fourth and fifth ribs in the axillary line. The outer portion of the tumor was removed, but the patient died of septic pleurisy and pericarditis due to an ichorous inflammation of the pedicle. At the autopsy the latter was found to be continued into a flattened subpleural lipoma as large as a hen's egg. Gussenbauer[3] examined a female patient, aged twenty-nine years, who had a tumor at the upper border of the left mammary gland. It was of the size of a nut and lay in the second intercostal space between the edge of the sternum and mammillary line, beneath the mammary gland and the pectoral muscles. The latter structures could be moved over the tumor which could not be displaced from the thoracic wall. Three years later the patient again was seen, the tumor having become enlarged in the mean time, and especially within the previous months. It was regarded as an encapsulated lipoma proceeding from the second intercostal space, on account of its mobility, consistency, lobulated surface and place of fixation. Its matrix was considered to be the subpleural fat tissue, as in the case reported by Czerny, which Gussenbauer also had seen, and in accordance with the previously mentioned statement of Rokitansky concerning the subpleural fat. At the operation a pedicle twice as thick as the finger was extended from the tumor into the chest through the widened second intercostal space. Traction upon the pedicle brought to view a portion of the intrathoracic tumor. The latter was enucleated by the finger from its subpleural attachments and was removed. It was a little smaller than the outer tumor, about the size of a man's fist, and was contained within a

[1] Wien. med. Woch., 1875, Bd. xxv. p. 166.
[2] Inaug. Diss., Halle, 1889.
[3] Loc. cit., p. 322.

subpleural cavity. The entire tumor was twice as large as the fist, and weighed over five hundred grams. The patient recovered.

The fat tissue of the mediastinum also is known to have served as the place of origin of intrathoracic lipomata which have projected externally from the wall of the chest, and have been submitted to surgical treatment. Cruveilhier[1] states that during an operation for the removal of a lipoma in front of the sternum the surgeon found several prolongations continued from it along the edges of this bone into the anterior mediastinum. Most of these were withdrawn and cut off. The patient died of suppuration of the anterior mediastinum. He mentions[2] also that Morel-Lavallée successfully removed from the sternal region the superficial portion of a lipoma which was continued into the anterior mediastinum by a prolongation which was not disturbed.

Kronlein[3] reports the case of a female infant, aged one year, operated upon by Langenbeck. At the age of six months a small tumor near the right breast was noticed by the parents. It grew rapidly, and at the time of operation formed a lobulated mass extending from the right axillary line to 3 cm. beyond the left sternal edge, and from two fingers' breadth below the right clavicle to the xiphoid cartilage. At the left of the tumor there was dulness on percussion preventing an accurate determination of the outlines of the heart. The apex beat was palpable in the sixth interspace, 2 cm. outside the mammillary line. The tumor increased considerably in size in the course of five days, and Langenbeck suggested its probable lipomatous character and congenital origin from the anterior mediastinum with subsequent perforation of the thoracic wall. On exposure of the tumor it was found to be continued into the chest through the third intercostal space, 1 cm. from the right sternal edge, by a pedicle as thick as the thumb. The pedicle was cut across at the level of the ribs, and the external tumor removed. The infant died of erysipelas. At the autopsy a rounded lipoma as large as a child's head, and covered with a firm, fibrous capsule, was found completely filling the anterior mediastinum, and continuous with the pedicle above mentioned. It projected 2 to 3 cm. beyond the right sternal edge, and filled almost the entire anterolateral portion of the left thoracic cavity, leaving only a small space below and behind, in which lay the compressed lung and the displaced heart.

The following unique case shows that subpleural lipomata may have a medical as well as a surgical interest. The patient, a fisherman, aged thirty-four years, lean, always well with the exception of a "typhopneumonia," entered the Massachusetts General Hospital January 4, 1905. He had been seized with a severe chill ten

[1] Loc. cit., p. 315. [2] Loc. cit., p. 322.
[3] Arch. f. klin. Chir., 1877, Bd. xxi.; Supplement, 157.

days previously, followed by pain across the chest aggravated on deep inspiration. There was a distressing cough, with rusty sputum and considerable dyspnœa. The physical signs indicated consolidation of the left upper lobe.

In the lower left axilla, corresponding to the region of the inferior third of the lower lobe of the lung, there were dulness on percussion and absence of respiratory and vocal sounds. The midaxillary region was occupied by a wedge of normal resonance and respiratory sounds, with its apex in the vicinity of the left nipple. There were dulness, diminished fremitus, and feeble vocal and respiratory sounds in the left back between the angle of the scapula and the spine. Numerous medium, moist rales were heard at the base of the left lung behind. The left border of cardiac dulness was four and three-quarter inches from the median line, and the right border of dulness was at the right sternal edge. The apex beat was audible in the fifth intercostal space four and one-half inches to the left of the median line. The heart sounds were regular, rapid, and distant; the second sound was strong at the apex. Adventitious sounds were not heard. There were 41,200 leukocytes per cubic millimetre and 80 per cent. of hæmoglobin.

During the subsequent four days the temperature fell from 104° to 101°, near which latter point it persisted. In the same period of time the pulse fell from 145 to 100, near which rate it subsequently remained. The respiration at first was from 50 to 35, but after five days ranged from 25 to 30, until within two days of his death, when it rapidly rose to 45. Death occurred two weeks after entering the hospital.

For a few days he was mildly delirious, the white count remained high, the signs of consolidation persisted, but on the fifth day the failure to improve and the faintness of the heart sounds suggested the possibility of an acute pericarditis.

The general condition of the patient then became somewhat better, but the physical signs persisted and the leukocyte count remained high. Attention was constantly directed to the pericardium, as well as to the lungs and pleuræ, in search of an explanation of the persistent elevation of temperature, and the continued leukocytosis.

Ten days after entrance and the twentieth day of the disease it was noted that the signs of consolidation continued in the left lung. The right border of cardiac dulness remained at the right sternal edge, the cardiohepatic angle was obliterated, and the heart sounds were faintly heard. The pulse became paradoxical. Although dulness persisted in the left back between the angle of the scapula and the spinal column, the vocal resonance and fremitus and the respiratory sounds were normal.

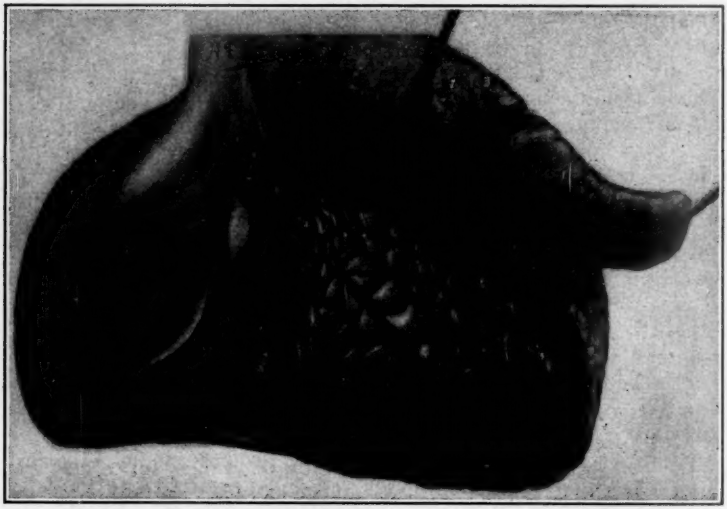
On the following day, the twenty-first of the disease, the pericardium was tapped in the fifth interspace near the left border of the sternum, and again in the left fourth interspace some two inches

from the sternal edge. Although the cannula entered at least an inch and a half in each place, and the surface of the heart was reached, there was no escape of fluid. Two days later, in consequence of attacks of dyspnœa and cyanosis, although there was no change in the physical examination of the chest, the exploratory trocar and cannula were inserted through the sixth left interspace in the axillary line into the subjacent area of dulness with the thought that pus might be present. The instrument entered readily between two and three inches and the tip was moved about freely. After withdrawal of the trocar no fluid escaped, nor did air enter the cannula. The trocar was reinserted and withdrawn and a second attempt at aspiration was made, but without effect. When the cannula was removed it was found to contain a fragment of soft material suggesting fibrin, but which, on microscopic examination, proved to be fat tissue.

My assistant, Dr. Beals, was asked to explore the pericardial cavity from the left xiphicostal angle after the patient's death, which took place the same day. He withdrew about a pint of creamy pus.

A postmortem examination was made by Dr. James H. Wright, from whose record the following abstracts are made: "On opening the pericardium a large amount, probably 500 c.c., of yellowish opaque fluid gushes forth. . . . Adherent to the inner aspect of the pericardial sac and to the surface of the heart everywhere is a dirty yellowish, finely shaggy or rough fibrinous layer 1 to 3 mm. thick. . . . The epicardial fat over the right ventricle near the pulmonary artery is much larger in amount than normal, forming a layer 3 or 4 mm. thick. . . . Attached to the pericardium on the left side and apparently continuous with the fat tissue of the superior mediastinum is a large mass of fat tissue of about the volume of a newborn child's head, divided by delicate connective-tissue septa into larger and smaller lobules. It evidently occupied the inferior and anterior portions of the left pleural cavity, the left lung being behind and above it. The inferior lobe is of about one-third the normal volume. . . . On section it is dark red in color, moist and not obviously atelectatic. The appearance of this lobe of lung suggests rather a hypoplasia than atrophy and atelectasis. The large amount of fat tissue just referred to is more or less adherent by fibrous adhesions to the diaphragm, to the pericardium, to the parietal pleura, and to the inferior lobe of the left lung. Portions of it were left in the body on removal of the thoracic viscera. As far as could be determined the shape of the mass of fat tissue was more or less that of a pear, the small extremity being attached most firmly to the external surface of the pericardial sac along a line extending from the anterior mediastinum in the region of the pulmonary artery (where it seemed to be continuous with the fat tissue of the pericardium) downward on the left side toward the posterior mediastinum. The

mass of fat tissue appeared to be covered in places by a thin, shiny layer resembling the pleura. The superior lobe of the left lung is voluminous and extremely resistant to the touch. On section the cut surface is grayish, granular, firm, and when scraped with a knife yields a rather thin, reddish fluid. The pleural surface of the superior lobe is grayish and opaque, not shiny. . . . Fluid of pericardium aspirated postmortem shows growth of pneumococci by culture. . . . Most of the air cells in the section from the superior lobe of the left lung contain an exudate consisting chiefly of fibrin in dense strands and masses. In a few places there is an outgrowth of fibrous tissue into the air cells, not sufficiently extensive to justify calling the condition organizing pneumonia.



Intrapleural lipoma proceeding from the fat tissue of the superior mediastinum.

The appearances in the section indicate a lobar pneumonia in which resolution has not been thoroughly established."

It is evident from the history of the patient and from Dr. Wright's examination that a pneumococcal pericarditis developed without characteristic symptoms or signs in the course of an acute pneumonia of the left upper lobe and that the resolution of the latter was delayed. Owing to the persistent solidification of this lobe and the presence of a large intrapleural lipoma of mediastinal origin the pericardial exudation was not enabled to cause a lateral increase of pericardial dulness. On the contrary, as was made evident by aspiration, the fluid was accumulated behind and below the heart.

The congenital origin of the lipoma is indicated by the accompanying hypoplasia of the lower lobe of the left lung. Its discovery

was wholly unexpected, although the physical signs and the result of aspiration would permit a diagnosis in a second case. Its significance to the physician lies in its presence being a satisfactory explanation for an unusual distribution of pericardial exudation, and its localization offers an opportunity of error in the diagnosis of a circumscribed pleuritic exudation.

It is a matter of common knowledge that even considerable quantities of liquid pericardial exudation occasionally escape recognition. Characteristic symptoms may lack and the heart adapt itself to the encumbrance of moderate accumulations of fluid, and, as a rule, the quantity present is small. Pericardial paracentesis, therefore, is of relatively infrequent occurrence. During a period of thirty-four years at the Massachusetts General Hospital there are noted in the index of the records 150 cases of acute pericarditis, of which 14 were tapped; 5 of these died, 4 were discharged much relieved, 2 relieved, and 3 well. In 4 cases no fluid was obtained, while in the remaining 8 quantities varying from one drachm to one quart were removed. In 6 cases less than four ounces were obtained.

Since October, 1896, evidence of acute pericarditis has been noted in twelve autopsies. The largest quantity of fluid found was nearly one pint, and in the patient concerned the existence of a pericarditis was not suggested until the day of death. In a second instance a half-pint was present, but in the majority of the cases the quantity of fluid observed was less than five ounces.

The diagnosis of an exudative pericarditis is determined by rational signs indicative of an infectious process and by physical signs calling attention to the seat and extent of the exudation. The rational signs alone are not to be depended upon to direct suspicion to the pericardium, since pericarditis usually is secondary to an inflammatory process elsewhere, for example, in the lung, pleura, or peritoneum, the symptoms of which are likely to mask those proceeding from the pericardium.

Especially significant in the etiology of the cases tapped in the hospital were acute pneumonia and rheumatism, each in four cases, while the pericarditis was regarded as primary in three instances. The diagnosis is comparatively easy when the course of the disease can be traced from the initial pain and friction sound to the gradually increasing area of cardiac dulness of characteristic outline, with fading rub and increasing remoteness of cardiac sounds. It is rendered more difficult when the adherent lungs prevent the lateral distention of the pericardial sac, the adjacent lung is solidified, the pleural cavity contains an exudation or the anterior wall of the heart is united to the parietal pericardium by old or recent adhesions. To these familiar obstacles a new source of difficulty is to be added—viz., the intrapleural lipoma, especially when it attains the size and position shown in the case here reported, as well as in that of Krönlein-Langenbeck.

The experience above recorded indicates the essentially exploratory nature of pericardial paracentesis and emphasizes the importance of a thorough exploration in doubtful cases. As usually performed, the needle is inserted into the fourth or fifth left intercostal space near the sternal edge or some two inches from it. The failure to obtain fluid at these points in the case here reported led to the assumption that the exudation was largely fibrinous. The absence of an increased area of dulness at the right of the sternum negatived the thought of tapping at Rotch's point in the fifth right interspace, one and one-half inches from the sternum. The positive result of the postmortem puncture in the left xiphicostal angle makes it obvious that this way into the pericardium should always be favored in cases of suspected exudation, if not in the first instance, certainly when aspiration at other points has proven ineffectual.

Larrey[1] originally recommended this route for pericardial drainage, and gave specific directions for performing the operation. He was led so to do from having successfully drained in this region a stab wound, which gave satisfactory evidence of communicating with the pericardial cavity. Roberts,[2] however, objected to paracentesis in this situation unless the case were exceptional, from the possibility of wounding the diaphragm and liver, and from the difficulty of determining the edge of the ensiform cartilage in fat persons. In two cases in which I have punctured the pericardium in the left xiphicostal angle, although fluid was not obtained, no ill effect followed, and the condition of the patients improved temporarily at least, after the exploration was made.

From the facts above narrated, and from those reported by F. C. Shattuck[3] and others, an exploratory incision in the xiphicostal region seems advisable in urgent cases when exploratory puncture has afforded no relief. Roberts[4] recommended exploratory incision as far as the pericardium when there was a doubt between a large pericardial exudation and a dilated heart. More recently he states[5] that incision is better than aspiration, even when the affection is not supposed to be purulent. Brentano[6] advocates in all cases pericardial incision with previous costal resection as less dangerous than paracentesis, and Weintraud[7] prefers incision to paracentesis. Although Roberts[8] now is in favor of exploratory aspiration in the upper part of the left xiphoid fossa, he considers that this region does not afford room for incision and drainage. Ogle and Allingham,[9] on the contrary, recommended the plan

[1] Clin. Chir., 1829, tome ii. p. 203.
[2] Paracentesis of Pericardium, 1880, p. 66.
[3] Transactions of the Association of American Physicians, 1897, vol. xii. pp. 193, 196.
[4] Annals of Anatomy and Surgery, 1881, vol. iv. p. 250.
[5] The American Journal of the Medical Sciences, 1897, vol. cxiv. p. 649.
[6] Deutsch. med. Woch., 1898, Bd. xxxii. p. 507.
[7] Lehrb. d. klin. Untersuch. Meth., 1905, Bd. ii. p. 380.
[8] The American Journal of the Medical Sciences, 1897, vol. cxiv. p. 652.
[9] Lancet, 1900, vol. i. p. 693.

of reaching the pericardium originally outlined by Larrey, followed by the removal of two inches of the seventh left costal cartilage, if necessary. Latham and Pendlebury[1] reported the successful performance of this operation with a vertical instead of an oblique incision and the removal also of two inches of the sixth costal cartilage, and Pendlebury[2] has operated upon two additional cases, one of which recovered.

Mintz[3] also has successfully drained the pericardium in the region recommended by Larrey.

There is, therefore, no doubt that aspiration through the left xiphicostal space has disclosed pericardial exudation which was not made evident by puncture elsewhere, and that successful drainage has been established through the same region. Consequently this route is to be recommended for exploratory aspiration, incision and drainage, since it gives ample room (by resection of the costal cartilage, if necessary), is free from risk to the pleura and peritoneum, and enables injury of the internal mammary vessels to be avoided or controlled. In critical cases when aspiration causes no outflow of fluid and the tip of the instrument can be moved about within the pericardium exploratory incision should speedily follow.

THE TREATMENT OF CHRONIC OBSTRUCTION IN THE LARYNX AND TRACHEA.[4]

By John Rogers, M.D.,

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The causes of chronic obstruction in the larynx and upper trachea are generally classified as follows:

1. Cicatrices of traumatic, operative, or pathological origin. When following tracheotomy the deformities may be due to (*a*) contraction of the posterior soft part of the trachea following separation of the anterior segments by the cannula; (*b*) dislocation of the cricoid forward and downward, especially when the fistula is through or close to it; (*c*) overriding of the cut anterior ends of the tracheal cartilages in too long a fistula; (*d*) inversion of the cut anterior ends of the tracheal rings, especially if the fistula is to one side of the median line; (*e*) contraction of the trachea around the cannula due to sloughing of the cartilages or rings; (*f*) spur formation posteriorly (as after enterostomy), especially in combination with contraction of the posterior soft portion of the trachea, or dis-

[1] Lancet, 1903, vol. i. p. 798.
[2] Ibid., 1904, vol. ii. p. 1145.
[3] Centralbl. f. Chir., 1904, Bd. xxxi. p. 59.
[4] Read before the American Laryngological Association, Atlantic City, June 8, 1905.

location of the lower segment forward and the upper backward, or both.

2. Granulations around a cannula or intubation tube which are much less common than is generally believed. They have been described as occurring at the upper anterior angle of the fistula for a tracheal cannula, or opposite the usual fenestrum on the posterior tracheal wall, or at the lower end of the cannula or around its whole length, or about the upper or lower end of an intubation tube.

3. Hypertrophic subglottic laryngitis most commonly encountered in retained tube cases, less often in those with a retained tracheal cannula and almost always after diphtheria.

4. Prolonged and repeated diphtheritic inflammation, which must be exceedingly rare in these days of antitoxin, although it is worth noting that there is at least one case on record in which the exudate was demonstrable on the 151st day.

5. Postdiphtheritic paralysis of the laryngeal abductors, which is sometimes described and must be very rare, but clinically is indistinguishable from the following:

6. Atrophy of the laryngeal abductors from disuse in retained tube and tracheotomy cases.

7. Obturation or compression of the respiratory tract by neoplasms is common and involves first the removal of the tumor and next the treatment of any concomitant or resulting cicatricial stenosis.

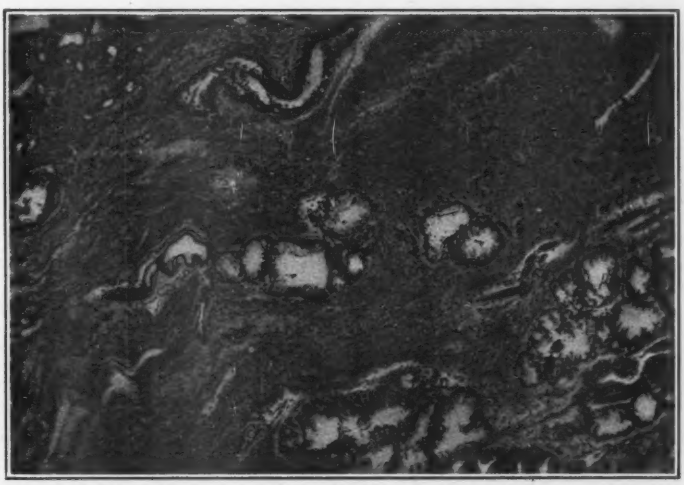
Of these "chronic hypertrophic subglottic laryngitis" needs some discussion. It was described by Köhl[1] as one of the common causes for retention of the tracheal cannula after acute diphtheritic stenosis long before the days of intubation, but at present is generally regarded with suspicion or mistaken for "granulations" or a cicatrix. I had the correct diagnosis first forced upon me by undertaking laryngotomies to remove these supposed granulations or cicatrices in cases of retained tubes. The gross appearance was that of a general velvety thickening of all the soft parts within the thyroid and cricoid cartilages, which bled easily, though there was no ulceration. The vocal cords and ventricles showed only as a slight tumefaction or dimple, and when the divided thyroid alæ were brought in apposition the hypertrophied mucous membrane completely occluded the laryngeal lumen. The futility of operation in such a condition is obvious and to elucidate its pathology, as clinically at least, it is one of the commonest causes of retained intubation tubes, I must repeat in full the findings I have been able to collect.

I am indebted to Dr. F. Tilden Brown for the first specimen, that of a child intubated at the Willard Parker Hospital June 28, 1896. Autoextubation, followed by closure of the larynx within a

[1] Archiv f. klin. Chir., 1887, Band xxxv. pp. 75 und 403.

few minutes, necessitated tracheotomy about a month later. A year subsequently, or on July 31, 1897, as the stenosis was unchanged, Dr. Brown inspected the larynx through a thyrotomy at the Presbyterian Hospital. There was an even thickening of the soft parts throughout the whole interior of the laryngeal cartilages. There was no sodden or watery appearance such as might be produced by œdema, but the thickened tissue seemed firm and vascular. Bleeding was easily excited by sponging. There was no sign of ulceration nor cicatrization, and the soft parts were apparently covered everywhere with epithelium. A vertical strip of tissue was then cut from the whole thickness of the soft parts on each side of the inner surface of the larynx. The pathologist of the hospital reported that

FIG. 1



these strips showed "infiltration with many small round cells, in places apparently granulation tissue. Over some thickened areas there is thin mucous membrane." It would be difficult to cut sections of such strips in a plane which would demonstrate the presence and integrity of the epithelium throughout, in other words, to prove by the microscope the absence of ulceration. But this may be inferred from the gross findings in spite of the use of the words "granulation tissue," which are intended to indicate a state of exudative and productive inflammation. This child died later of pneumonia.

The operation did not succeed in making respiration possible without the tracheal cannula.

I am indebted for the second case to Dr. Dillon Brown. This

child was intubated by him for laryngeal diphtheria in the first week of February, 1897. The disease subsided, but the stenosis

FIG. 2

persisted and necessitated "retention" of the tube. Respiration without it was barely possible for about an hour, but no longer, and finally after autoextubation at the end of a month from the time of

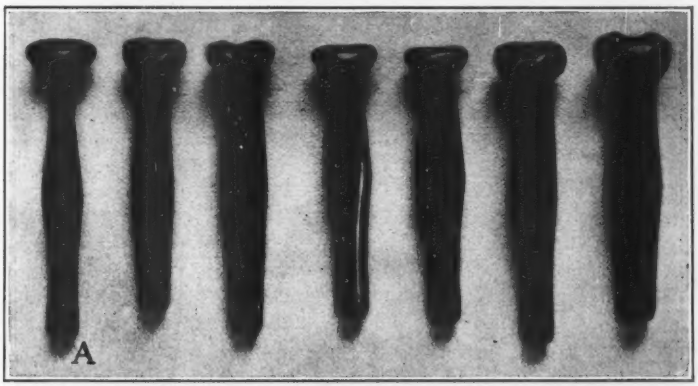
FIG. 3

first insertion asphyxia occurred. At autopsy the intralaryngeal soft parts were found to be evenly thickened to such an extent that

the lateral portions of the larynx were in contact in the median line. The tissues were moderately soft and compressible enough to permit the introduction without undue force of the tube ordinarily employed for a child of this age and size.

Dr. W. N. Berkeley, the pathologist then in Dr. Brown's laboratory, cut some sections for me transversely through the centre of the larynx and made the following report: "The mucous membrane, submucosa, and remaining soft parts within the larynx appear much swollen and thickened by a growth of new cells. The vessels are numerous and congested. There is an irregular increase in the superficial epithelium and an hypertrophy of the subepithelial papillæ. There is no evidence of ulceration with loss of substance. The striated muscle fibres, both in front and behind the arytenoid cartilages, are present in the usual amount, but like all the other soft tissues, are infiltrated with columns of small round cells. In all probability this infiltration would interfere with or abolish the function of the muscles. The cartilage is normal."

FIG. 4



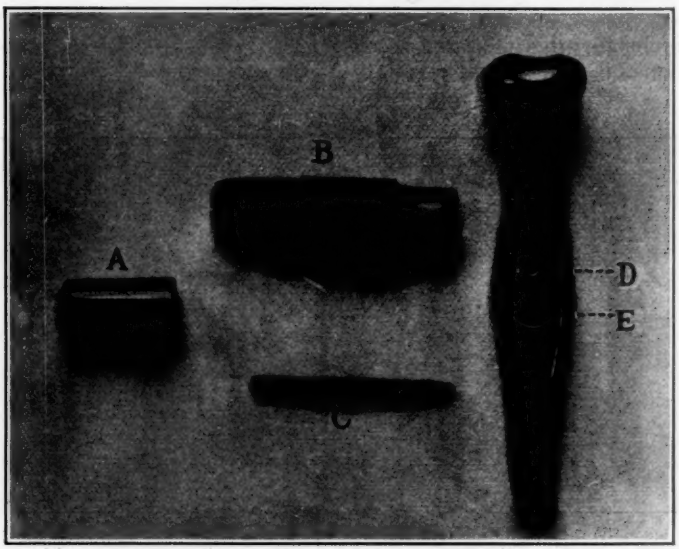
The third specimen is from Case VI. and is one approaching a cure. This child had worn an intubation tube for nearly three years and had never been tracheotomized. Only during the latter two months of tubage (and her life) was natural respiration possible for even a few hours.

The gross appearance was one of smooth, velvety thickening of all the soft parts within the laryngeal cartilages. This thickening reduced the lumen of the larynx to about one-third of its natural size. Within the anterior portion of the cricoid there was an irregularly circular area about one-quarter of an inch in diameter, which was evidently a cicatrix resulting from an ulcer, and another similar area an inch farther down and opposite the lower end of the tube

in the anterior and lateral portions of the tracheal mucous membrane about one-half inch in diameter. These areas did not constrict the lumen of the larynx or trachea, but, on the contrary, seemed slightly depressed below the surrounding soft tissue. Ulceration had therefore occurred at these points and the healing had increased rather than decreased the lumen of the passage.

To make clear the explanation of the treatment proposed I must anticipate by stating that by a "normal" tube is meant one of the ordinary O'Dwyer instruments as originally made by George Ermold & Co. in seven sizes, usually distinguished as suitable respectively for a child one year old or younger—two, three, four to five, six to seven, eight to nine, and ten to twelve years of age. For adults the

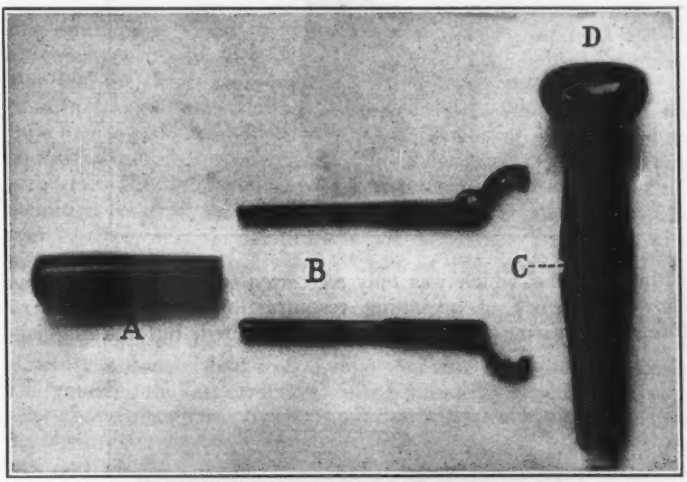
FIG. 5



sizes run from one to ten inclusive. By "special" tube is meant one of the same length as the normal, but in which the neck or constricted portion just below the head or the part below the retaining swell is greater in the transverse diameter generally by $\frac{1}{32}$ inch or some multiple of this amount. The "retaining swell" or bulging central portion of the tube must at the same time have a transverse diameter at least $\frac{3}{32}$ inch greater than the neck or the tube will certainly be coughed out. In cases of hypertrophic laryngitis which are totally unable to breathe without a tube this difference is too slight and a difference of $\frac{4}{32}$ or even $\frac{5}{32}$ inch may be required. Life will often depend on this $\frac{1}{32}$ inch.

By a "plugged" tube is meant one fitted with a plug made to screw accurately into a perforation in the anterior wall of a normal or special tube through a tracheal fistula. The plug is perforated at its inner end by a hole which exactly corresponds in size with the lumen of the tube. The plug is provided with a retaining pin, which, when the plug is in place, prevents it from rotating and so obstructing in the slightest way the lumen of the tube. By previous measurement the tube has a hole bored in its anterior wall, which when the tube is inserted lies opposite the tracheal fistula. Into this hole the plug is then screwed and "pinned" at right angles to the tube. A cap is then screwed down over the outer end of the plug

FIG. 6

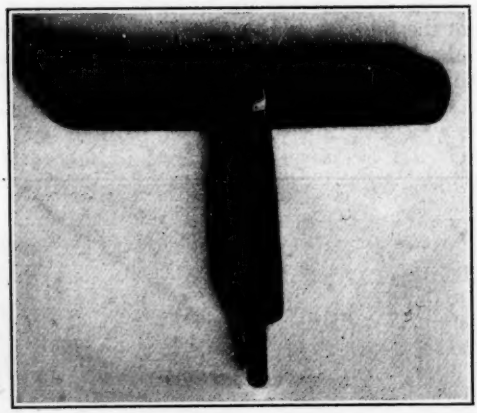


to hold the pin in place. By a "clamped" tube is meant one fitted with a detachable pair of metal clamps made on the principle of an obstetric forceps. In the sides of the tube are cut grooves, which, when the instrument is in place, lie at the level of the tracheal fistula. Through this the clamps are made to grasp the tube and are then held in place by a collar screwed down around them until the collar touches the tube. The plugged or clamped tube is a guarantee against autoextubation and at the same time keeps the tracheal fistula from closing.

The statistics of the 22 histories which could be submitted in full, did space permit, include 3 deaths, one from nephritis and pneumonia, 1 from autoextubation and asphyxia, 1 from asphyxia due to the clogging of a metal tube; 2 failures to cure; 7 still under observation. I do not consider a case cured until at least a year has

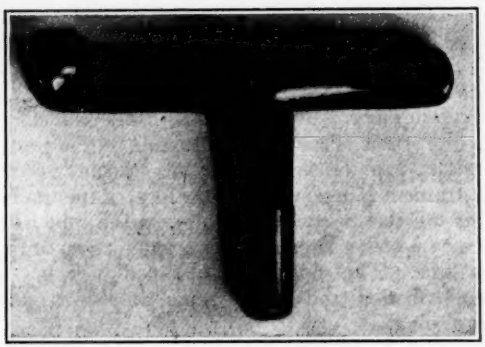
elapsed after the last intubation without the appearance of any signs of stenosis. Of these 7 cases: 3 are able to breathe naturally for a few hours or days, although the obstruction when first seen was complete; 2 (adults) have been from one to three weeks

FIG. 7



without a tube, which was only reinserted when the laryngoscope showed evidence of beginning recontraction (these 5 are therefore still wearing tubes); 2 apparently cured, but the requisite

FIG. 8



year has not yet passed; 10 cured, that is, they have remained free from any obstruction for at least one year and some for four or five years. All of these have voices which range from perfect to hoarse.

CASE VI.[1]—*Stenosis due to hypertrophic laryngitis following intubation persisting unchanged for two and a half years; some improvement during the next six months with "special" rubber tubes; adductor spasm evident; more marked improvement with special metal tubes; death ultimately from nephritis and pneumonia.*

G. S., aged three years, was intubated for diphtheria at the Willard Parker Hospital in July, 1902. The stenosis persisted and the tube was worn continuously thereafter with frequent changes until February, 1904. On this date a special rubber tube, of the four to five length, was inserted, having the neck and retaining swell one-thirty-second of an inch larger in diameter than the normal measurement. During the change absolutely no air passed through the larynx, no bleeding was produced, and no force was required for intubation. The condition was therefore clinically one of chronic hypertrophic laryngitis. February 20th the tube was coughed out for the first time in her history and also for the first time in two years she breathed three days unassisted. Then the tube had to be reinserted on account of increasing dyspnœa. This comparatively slow recurrence of the stenosis often occurs during recovery. It is too rapid to prove an error in diagnosis or that the condition is cicatricial. February 28th a larger special rubber tube was inserted, and special tubes of increasing size were used with infrequent changing until July 8th. At this change, when the largest of the special tubes had been worn a month, the condition seemed as bad as ever, and acting under the belief that a spasm of the adductors of the larynx might have something to do with the difficulty a normal four-to-five tube was inserted until July 16th. On removing the last or normal tube I found the breathing, though somewhat difficult, was fairly good and hence the supposition that the obstruction at the last change was due to a cramp of the adductors and atrophy of the abductors seemed correct. I therefore returned to the special metal tube, which had produced the first improvement in March. July 27th this tube was removed and cleaned by the house surgeon, as it seemed to be foul and producing a cough. Respiration, though difficult, was possible. In August a nephritis developed and this was followed by a chronic pneumonia, which at autopsy was pronounced by the pathologist to be simple and not tuberculous. August 26th the tube was removed and natural respiration for the first time was easily possible, but the dyspnœa gradually returned and on August 29th the tube had to be replaced. The child continued to gradually lose ground and finally died from the pneumonia on September 12th. The tube was not removed until after death, but then the larynx appeared

[1] The twenty-two histories which it was the author's intention to submit in detail made this article so long that at the request of the editor only those are presented which give some idea of the worst difficulties and the method of treatment. In consequence these convey a more gloomy impression of the prognosis than is warranted by the majority of the histories.

open enough for respiration to have been carried on unassisted. The report on the specimen will be found in the first part of this paper.

CASE VII.—*Stenosis due to hypertrophic laryngitis following intubation for laryngeal diphtheria; complicated by cicatrices resulting from several laryngotomies and tracheotomies; obstruction from spasm of the adductors; also from granulations; cured by two and a half years of treatment with "special" tubage; duration of stenosis, six years.*

M. M. was admitted to the Willard Parker Hospital June 2, 1896, at the age of three years. She had been intubated six days previously for diphtheria. The stenosis persisted and on account of frequent and dangerous autoextubation she was tracheotomized July 13th. The stenosis still persisted and intubation was again tried. Astringents like alum applied on the outer surface of the tube to the laryngeal mucous membrane, as advised by Dr. O'Dwyer and afterward curetting the larynx through the mouth, were tried without success. In October, 1896, transferred to the Metropolitan Throat Hospital and thyrotomy performed by Dr. Wagner. Some thickened tissue removed from the interior of the larynx and the wound packed. Breathing through the tracheal cannula. The only pathological change noted during the operation appeared to be a general thickening and hypertrophy of all the intralaryngeal soft parts. In October, 1898, another thyrotomy, performed by Dr. Hope, with division and excision of numerous cicatrices. In January, 1900, the patient, then seven years old, was transferred to Gouverneur Hospital still breathing through a tracheal cannula. Under chloroform a much deformed larynx, which barely admitted one of the smallest urethral sounds, was dilated from below through the tracheal fistula with urethral sounds up to No. 25 F. There was a "spur" opposite the fistula and the entrance to the trachea below was contracted around the cannula; this was also dilated like the larynx. A ten-to-twelve normal intubation tube was then inserted and subsequently changed every four or five weeks for cleansing. In May, 1900, the tracheal fistula finally closed and so cut off all respiration except through the larynx and the stenosis was still marked. At this change I made several attempts at intubation and failed on account of the deformity and displacement of the larynx due to the numerous cicatrices. The consequent traumatism produced complete obstruction and asphyxia was barely avoided by a hasty tracheotomy, after which intubation was finally accomplished. In July, 1900, as a preliminary to changing the tube and to avoid the previous danger, I again punctured the trachea and then intubated without difficulty. This largest-sized tube was changed at intervals of four weeks without perceptible gain and therefore with the object of obtaining the utmost dilatation a "special" tube was employed of the same length as the normal but of

much larger diameter. This was worn with the usual changes until January 15, 1901, when it was removed and a cure seemed at last obtained, but five days later at night there was a sudden attack of dyspnœa and the child was barely saved from asphyxia by another hasty tracheotomy by the house surgeon. Some infection followed with recontraction of the cicatrices, and not until March was intubation again practised. A little later dyspnœa began to reappear with the tube in place. An exploration revealed exuberant granulations at the base of the epiglottis overtopping the tube and blocking its lumen. In July another thyrotomy had to be performed to gain access to the obstruction and the granulations were then scraped and cut away. A normal tube with a small head to avoid pressure on the ulcerated surfaces was inserted at the close of the operation. This was worn until no more bleeding occurred at the periods for cleansing. In November the special tube with the large diameter was again used and worn with occasional removals for cleansing until February, 1902, when a cure seemed obtained, but that night there was a sudden attack of dyspnœa and another tracheotomy by the house surgeon. On the following day the large calibred special tube was reinserted and worn until May. Then a normal tube of the eight-to-nine size was substituted for two weeks, and in June the patient was at last dismissed cured. She has remained well and free from dyspnœa ever since and even has a good though husky voice. She is at present attending school (October, 1905).

CASE IX.—*Stenosis due to hypertrophic laryngitis following intubation for laryngeal diphtheria; thyrotomy followed by sloughing of the cartilages; permanent cure after three months of proper treatment.*

I. B., aged four years, admitted with diphtheria to Willard Parker Hospital and intubated in December, 1896. The stenosis persisted and necessitated retention of the tube. Frequent and dangerous autoextubation began, and to prevent asphyxia tracheotomy had to be performed on February 20th. A month later intubation with a normal four-to-five tube was practised with the tracheal cannula in place. No autoextubation occurred, so the cannula was removed and the fistula allowed to heal. The complete stenosis persisted and she was transferred to Gouverneur Hospital October 15, 1897. A week later the tube was coughed out and a hasty tracheotomy performed by the house surgeon. In November the cannula was removed and intubation again continued. January 10, 1898, as respiration was impossible without the tube, I explored the larynx by a median vertical incision through the thyroid and cricoid cartilages. The tissues were thickened and soft throughout the whole interior of the larynx, in which there were no ulcerations and no cicatrices. By bringing the parts together it was found that the hypertrophied mucous membrane completely occluded the lumen of the larynx. With the idea of producing a vertical cicatrix on

each side of the larynx and so retracting the swollen parts I passed a silk suture through the cartilage and thickened mucous membrane on each side and tied it on the outer surface of the corresponding thyroid ala. This retracted the tissues sufficiently to allow easy breathing and the larynx was closed, but infection followed and in the course of the next few weeks apparently the greater part of the thyroid and cricoid cartilages sloughed out. April 20th the patient was discharged seemingly in a hopeless condition and condemned to the permanent use of a tracheal cannula. In August the patient returned with a dense cicatrice occupying the position of the larynx, but under chloroform with some difficulty a probe was passed from the tracheal fistula up into the mouth. This was followed with sounds of increasing calibre until the larynx and the entrance to the trachea had been dilated up to No. 24 F. A normal four-to-five tube was then inserted, the cannula removed and the fistula allowed to close. On October 1st the tube was removed and a six-to-seven normal tube substituted. The stenosis was still nearly complete. At the next change, in November, natural breathing was found possible and the tube was left out. For the first two weeks there were several attacks of inspiratory dyspnœa, especially at night, but under the administration of a little morphine these gradually disappeared. Since then the cure has remained permanent and the voice is fairly good.

CASE XV.—*Stenosis from cicatrices of syphilitic origin; clogging of a metal intubation tube; extreme subsequent contraction of the larynx which required laryngotomy to insert a plugged tube; apparent cure followed by apparent failure owing to unrecognized spasm of the adductors.*

C. McD., aged twenty-five years, was admitted to Gouverneur Hospital August 18, 1896, suffering from marked laryngeal dyspnœa, which had been increasing gradually since the previous April. There was an indistinct history of syphilis. She was immediately tracheotomized and, as is usual in emergency cases, the cricoid cartilage was divided. Subsequent examination revealed a larynx much deformed and obstructed by cicatrices. There was no ulceration. On November 15, 1898, under chloroform the larynx was dilated up to No. 35 F., with urethral sounds entered through the trachea and a metal tube inserted. Much difficulty in swallowing followed and a week later I arrived just in time to extract the tube, which had become nearly occluded by dried mucus. Another intubation was refused and the tracheal cannula had to be inserted. Attempts to use a Dupuis' segmented right-angled cannula failed, owing to the difficulty in swallowing and to the pain and swelling in the top of the larynx over the upper end of the superior half of the instrument. There was the same difficulty with an ordinary curved cannula, to which was fitted on its upper surface a bivalve dilating attachment to extend up into the larynx. Intermittent dilatation

with Schroetter's tubes proved useless, as is usual in these bad cases. The patient left wearing a tracheal cannula. In February, 1899, she returned and under chloroform the larynx was found almost but not quite occluded. The thyroid and remains of the cricoid cartilage were then split in the median line upward from the cicatrix and the tortuous line of mucous membrane representing the remains of the lumen of the larynx was laboriously followed with a fine probe, as in external urethrotomy without a guide, until the pharynx was opened. A small adult intubation tube was then inserted through the mouth, and after making longitudinal incisions in the most prominent cicatrices the wound was sutured over the tube and a tracheal cannula placed in the lower angle beside the tube. A couple of weeks later an intubation tube with its anterior half cut away to receive the inner end of the cannula was attempted, but clogged with dried mucus and pus so often that it was abandoned and a "plugged" tube was substituted. This worked fairly well, but the fistula in healing came to be located about the middle of the thyroid cartilage. It, however, brought the tube near the surface and the patient could clean it daily with a feather after removing the plug. A change of the tube every four or five weeks demonstrated rapid improvement, and in May the tube was removed; the fistula in the neck contracted almost immediately, so that at the end of twenty-four hours it was practically closed. Meanwhile natural respiration was carried on without the slightest difficulty, but suddenly three or four days later, at night, an attack of dyspnœa almost caused asphyxia (adductor spasm), but fortunately enough of the fistula remained to enable the house surgeon to dilate it and insert the cannula. Further treatment was then refused for some time and the patient went home wearing the tracheal cannula. April, 1900, under chloroform, the contracted larynx and trachea were dilated as before. But so much difficulty in intubating was experienced on account of the "spur" which had formed in the posterior wall of the trachea opposite the fistula, that the attempt had to be postponed until a small right-angled blunt retractor, grooved on its convexity to fit the intubation tube, could be constructed. A week later, under chloroform, with this instrument in the lower angle of the fistula to drag the trachea forward and to guide the tube in the convexity of the retractor into it, intubation was accomplished. The tube used was one with a "plug" attachment. This was kept in place with occasional changes for cleansing for about four months or until August, 1900, then a cure seemed obtained, but two days later the fistula, having nearly closed, there was a sudden attack of dyspnœa as before at night and the cannula was reinserted and the patient refused further treatment. When seen last about a year later she was pregnant and showed signs of advanced pulmonary phthisis and has probably died since then, but at that time, though the cannula was still worn, laryngeal res-

piration, much to my surprise, was fairly good and another course of intubation would probably have produced a cure. But it seemed hardly justifiable in her condition. The apparent lack of success after prolonged dilatation was undoubtedly due to my failure to recognize and allow for the "cramp" of the laryngeal adductors. A tube with a small neck worn for a couple of weeks after the removal of the large ones would have permitted the abductors to regain their strength and so prevented the cramp or spasm of the adductors. She was a very nervous woman, somewhat addicted to beer and easily terrified, so that the sudden dyspnœa after apparent success would make her refuse trust in anything but the cannula. In the light of later experience I have regarded this case as really a success, though apparently a failure.

All the credit for the main principle in the management of chronic cicatricial laryngeal stenosis, that of long-continued dilatation with an intubation tube, of course belongs entirely to the late Dr. O'Dwyer. He long since insisted that this method was the only one generally applicable, and he published several cases of chronic stenosis due to one or another cause which were brought to a satisfactory conclusion by intubation continued for several months. But he had no regular course of procedure for curing the occasional cases of "retained tube" with chronic hypertrophic laryngitis, which in addition are often complicated by dangerous autoextubation, nor did he go into the details of the management of the various conditions accompanying stenosis with a retained tracheal cannula, and in these small particulars the histories of the cases given above supply much that is valuable. For there is no more serious disability than chronic respiratory obstruction in the upper air passages, and any method of treatment which is safe and offers a reasonable prospect of cure in nearly every instance should be welcome. Great patience, perseverance, and foresight with some technical skill are required, but given these my experience has led me to believe that a failure to ultimately restore natural respiration and even voice must be exceptional. Intermittent dilatation with Schroetter's instruments and all the various operative procedures which from time to time have been recommended chiefly in German literature are notoriously unsatisfactory. Some conditions must, however, occasionally be encountered which can only be remedied by plastic or other more or less extensive surgical interference, but they are rare and even then subsequent cicatricial contraction will probably arise. Primary union in the several laryngotomies mentioned above was much less common than suppuration, which may vary from simple failure of the wound to unite by first intention to extensive cellulitis of the neck with lymphadenitis and sloughing of the cartilages. In short, operations for stenosis of the larynx or upper trachea are seldom advisable except for the removal of tumors. Papillomata are probably the most common of these, and several

cases have been reported in which intubation alone was said to have caused their disappearance. But without the definite knowledge of the extent of the disease only to be gained by a previous thyrotomy or tracheotomy intubation may prove dangerous; the papilloma or any other growth may be caught in the point of the tube and masses of this tissue pushed down ahead of it into the trachea like a gun wad. Several years ago I encountered a death from asphyxia in the practice of a colleague produced in this way. Hence, of course, intubation, like any other surgical procedure, cannot be indiscriminately employed, and an open operation, if only to make sure of the conditions, may sometimes be a necessary preliminary. But aside from the inspection or removal of new-growths the causes of stenosis rarely require anything beyond dilatation followed by intubation.

These cases can therefore generally be grouped as obstruction due to (*a*) cicatrices, (*b*) hypertrophic laryngitis, (*c*) granulations or œdematous ulcer, and (*d*) spasm of the adductors. One or more of these conditions may coexist and before proceeding to the treatment it is necessary to differentiate briefly the symptomatology. A cicatrix is easily recognizable by the history of a traumatism or disease with or without ulceration, coupled with inspection and instrumentation of the larynx, or tracheal fistula if one has been made. The deformities common after tracheotomy have already been described. The chief difficulty lies in differentiating between some of the tumors, including the granulomata and the persistent stenosis—hypertrophic laryngitis—sometimes following intubation for an acute condition, generally laryngeal diphtheria. The tumors, of course, if they cannot be removed through the mouth, require open operation any way, and exploration is generally safer than working in the dark. But the differentiation of a cicatrix from the other common conditions should be simple even when an open ulcer overlies it. In the first place it offers considerable resistance to instrumentation, and after dilatation or intubation the stenosis returns gradually within days or weeks. An hypertrophic laryngitis offers no resistance to the insertion of a tube, and after its withdrawal the recurrence of the obstruction is immediate and often complete. When these two difficulties occur together, as is not infrequent, especially after diphtheria, the history coupled with the resistance to instrumentation and the sudden recurrence of dyspnœa after it, should present no great difficulties, although any traumatism of the larynx may give rise to an œdema of quite sudden onset. The spasm of the adductors, though not strictly speaking a cause of chronic stenosis, so closely simulates clinically an hypertrophic laryngitis that it must be included in the differentiation. It seems to have been recognized long before the days of intubation and so must occur whenever the laryngeal muscles have been for any length of time at rest. Of course, when an intubation

tube has been worn for a considerable period, particularly the "special" instrument which I recommend with an abnormally large neck, the abductors are put out of employment and must undergo an atrophy of disuse while the adductors are under some more or less constant tension with every attempt at coughing. However the explanation may be made, clinically at least there is no difference appreciable between spasm of the adductors and hypertrophic laryngitis after removing the intubation tube or plugging the tracheal fistula. When, however, this condition is suspected in a patient who has been intubated the diagnosis can sometimes at least be made by extubating under an anæsthetic. This removes the element of fright and abolishes the disturbances somewhat vaguely termed "reflexes," and normal respiration may be discovered where it might not otherwise be anticipated. Furthermore, a sudden spasm, and that, too, of very dangerous dyspnœa, generally occurring at night with coughing, and inspiratory rather than expiratory, can pretty safely be ascribed to this cause. The resistance encountered in this condition by the tip of an intubation tube catching on the closely approximated vocal cords should not be misinterpreted as a cicatrix, which takes time to develop and contract. The treatment of the two conditions is very different. "Granulations," if not perceptible to sight or touch, bleed easily upon instrumentation and generally occur as the result of the pressure or irritation of an intubation tube or cannula, in which they produce a more or less gradually increasing obstruction either to inspiration or expiration, seldom to both acts in succession. A spasmodic cough accompanied by more or less hemorrhage and dyspnœa may also give some indication of their presence; but I am convinced that they are a far less frequent cause of chronic obstruction than seems generally to be believed. When there is bleeding accompanied by stenosis following extubation or decannulization it is much more reasonable to ascribe the dyspnœa to a simple œdematous ulcer overlying an hypertrophic laryngitis or a cicatrix and not to exuberant granulations. The slight traumatism incident to the relief of the pressure of an intubation tube against such an ulcer or the "gagging" which often accompanies the removal of a cannula, especially if it is high up, can produce enough congestion in an eroded area to cause it almost immediately to nearly or quite occlude an already narrow space. I have never been able to positively verify such a condition but believe in its occurrence, and though it may be impossible to differentiate from a pure hypertrophic laryngitis, which is also very vascular and bleeds at the slightest touch, the treatment does not differ.

In a former article on this subject I recommended the long-continued use of as large a normal intubation tube as the larynx would admit, but experience has caused me to modify this treatment very materially for the different conditions. Simple chronic hypertro-

phic subglottic laryngitis is probably the most common cause of obstruction after intubation. This ought to read "after diphtheria," because it seems unfair to imply that it is due to intubation inasmuch as it was recognized as one of the most frequent obstacles to the removal of a tracheal cannula long before intubation was invented.[1] It may also occur entirely independently of any acute disease, and Case I. seems to be an example of this. There will, however, be considerable difficulty in convincing most intubators that unskilled or rough instrumentation does not have something to do with its inception or continuance, for a certain number of these cases will get well in the course of days or weeks with careful and frequent change of a tube, smaller if need be than the development of the patient would ordinarily indicate, the object being to minimize traumatism. Local astringents, as advised by Dr. O'Dwyer, applied to the interior of the larynx in a coating of gelatin or other substance adhering to the outer surface of the tube, may also succeed. But there are a number of cases (about 1 in every 100 intubated as estimated from the statistics given in my previous paper[2]) which will resist all these efforts for their relief. If the stenosis of this character has persisted for about a month in spite of all the usual plans of treatment, and spasm of the adductors can be excluded, another method must be adopted. Some of the patients will recover if as large a normal tube as can pass the cricoid cartilage is used for five to six months and changed at intervals only for cleansing. At the end of every four or five weeks the tube becomes foul and causes a bad odor in the breath and considerable cough. But usually in a persistent case it is better to start with a "special" tube or one having the neck one or more thirty-seconds of an inch larger in the transverse diameter than the normal instrument, with the object of applying pressure upon the thickened area within the larynx. As this area generally extends from above the vocal cords to or through the cricoid cartilage, and as the retaining swell of the tube must be enlarged with the "neck" to prevent autoextubation the retaining swell of the special tube must be enlarged along with the neck; and the same must be done with the head or there is danger of the instrument slipping below the epiglottis, which will then more or less completely occlude the upper aperture. Practically in these cases I am accustomed to make trials with normal tubes of increasing calibre under complete anæsthesia if necessary, until I have found the largest tube which will enter and pass through the larynx with some slight force. The "special" tube is then made of the length of the normal tube proper for the age and development of the patient, but with a retaining swell having the diameter of that of the trial tube. The neck must then be at least three-thirty-seconds of an inch smaller than the retaining swell and the head six-thirty-

[1] Kühl. Archiv. of klin. Chir., 1887, Band xxxv. p. 75.
[2] Annals of Surgery, May, 1905.

seconds of an inch larger than the retaining swell. If a metal tube is to be used its weight is better and more safely sustained by a head which is at least seven-thirty-seconds of an inch larger than the retaining swell and a large tube should have eight-thirty-seconds of an inch difference. If the child is under four or five years of age and so needs the smaller sized tubes the thirty-seconds of an inch difference should be altered to sixty-fourths of an inch. Moreover, a difference of three-thirty-seconds of an inch between the neck and retaining swell is the minimum difference which I have found by experience sufficient to keep the tube in place. Some patients will require four-thirty-seconds or even five-thirty-seconds of an inch difference. In the first part of the treatment of an hypertrophic laryngitis, especially if the stenosis is complete, as is frequently the case, it is wise to start with a special tube having a diameter of the neck five-thirty-seconds of an inch less than that of the retaining swell. Care in these seemingly minute details may mean all the difference between life and death, as unexpected autoextubation or aspiration of the tube into the trachea (impossible with the normal tube) can easily be fatal accidents. After a special tube thus carefully made has been worn a month or six weeks it usually must be changed and cleaned or the accumulation on it may cause it to clog suddenly, or produce ulceration, or a septic bronchitis, or pneumonia. Another tube should be at hand of one-sixty-fourth or one-thirty-second of an inch larger diameter through the neck and retaining swell than the last. Some slight improvement in the stenosis can generally thus be detected in the resistance offered to intubation if it is not clinically apparent. The judgment of the operator must determine the amount of presure which can be borne without sloughing. Fortunately the larynx seems capable of bearing much more traumatism in these chronic conditions than one would think possible, and if some little ulceration occurs as it often must, the epithelium will in course of time cover the raw area unless the pressure is extreme. Furthermore this ulcerative process, if not too extensive, may actually be the way in which cure takes place in these cases of simple hypertrophic laryngitis. Scattered areas of fibrous cicatricial tissue may in contracting against the cartilages do away with the preceding chronic exudative and productive inflammation. A most important question in these and to a less extent in cases having another etiology lies in the use of metal or vulcanized rubber tubes. The metal instrument is usually made of bronze or some less expensive compound heavily gold plated. At any rate it is composed of two different metals, the exposed part being gold. In two or three of the cases of hypertrophic laryngitis described above absolutely no progress could be made after weeks or months of treatment with hard rubber tubes so constructed as to make all the pressure the larynx seemed able to bear. Then after the substitution for the special rubber tube of a similar or

slightly smaller compound metal tube, either a cure or very marked improvement was found at the next change or within four or five weeks. I am unable to explain this fact except on the theory of some electrolytic action. The metal instrument is far heavier than the rubber and the pressure may have something to do with its advantages, but I doubt it, as I know I have made more pressure with a large rubber than with any metal tube and there must be some other reason for the difference. The claims for electrolysis are generally regarded with suspicion, but these metal tubes, after being worn a month or two, become heavily coated wherever they press most upon the larynx, that is chiefly around the head and neck, with a stony, calcareous deposit, and under and around this the gold plating entirely disappears. There is of course no acid or alkali in the body which can dissolve gold, and it does not chip off but apparently dissolves. The external calcareous deposit of course roughens the surface, and if the tube is worn for a long time may reach such proportions as to prove a very serious obstacle in extraction. I once met with such a case in the practice of a colleague in which the tube, having been in place for several months, required for removal general anæsthesia and a strong pair of forceps to permit a firm grasp of the head of the tube. The metal tube will, however, if so constructed as to exert moderate pressure upon all the thickened tissue of an hypertrophic laryngitis, sooner or later produce a cure. But the patient who wears one must be constantly watched, as it has a tendency to clog and thus asphyxiated one of my cases and very nearly did the same with another (Case XV.), and naturally after those experiences I have always been afraid of these tubes and only resort to them when the special hard-rubber instrument after thorough trial in increasing sizes proves a failure.

Autoextubation is a frequent and very dangerous complication in any intubated case, but especially dangerous in hypertrophic laryngitis and introduces the "special," or normal "clamped," or "plugged" tubes. Some patients will cough out any tube which has not such a mechanical attachment to keep it in place, and as autoextubation is most common with hypertrophic laryngitis, I have come to believe that some degree of this condition is, if not the primary, at least the secondary cause of the difficulty. The chronically inflamed tissues are irritable and their contact with the tube excites coughing accompanied by retching or involuntary expulsive contraction of the pharynx. The combination of an attempt to cough and vomit at the same time seems the surest way for the patient to get rid of the tube, and life generally depends on its immediate reinsertion or a hasty tracheotomy. The latter operation probably, by giving rest to the larynx, seems occasionally to produce a cure, but ordinarily it only adds a cicatrix and a possible tracheal deformity to an already bad-enough condition. If there were no other way to escape the dangers of autoextubation it might

be justifiable, but hypertrophic laryngitis used to be one of the most frequent obstacles to decannulization and it seems hardly proper to inflict a tracheal fistula for its cure. Still, the tube must be kept in the larynx and for this purpose I offer the "clamped" or "plugged" instrument. If autoextubation is at all frequent the trachea is carefully opened in the median line through the second and third rings and the intubation tube while in position marked to correspond with the incision, or if a tracheal fistula has already been made the tube is inserted and marked at a point coinciding with the fistula. During the few succeeding days required to make the instrument the cannula will have to be worn, but this inflicts the least possible damage. Some explanation is required as to the comparative advantages of a "clamped" or "plugged" tube and as to the use of such an instrument in general. It is assumed that the trachea is opened carefully in the median line well below the cricoid, and only enough to comfortably admit the cannula and subsequently the retaining attachment for the tube. Deviations from these rules as indicated previously promote cicatrices and tracheal deformities. If the fistula is already existent when the patient comes for treatment, as is frequently the case, there is of course nothing to be gained and much may be lost by enlarging or altering the position of the fistula, and it is then only a question of a normal or "clamped" intubation tube. The self-retaining instrument is far preferable to a tracheal cannula, to which it is the only alternative in hypertrophic laryngitis complicated by frequent and dangerous autoextubation if only because it places the patient in a practically normal condition with the single exception of a whispering voice. For less serious cases of stenosis, especially those which experience has demonstrated can breathe for an hour or two without mechanical assistance, the self-retaining tube may be thought to possess doubtful advantages. But in these there are many factors to be considered. To be sure at least one of the patients in this series could not swallow while wearing the instrument, but that experience is very exceptional, and in adults, especially "nervous" subjects, equally common with the normal tube. That the clamp or plug may act like the cannula in promoting or maintaining a tracheal deformity or favor a necrosis of cartilage has always been my fear, and hence I never use it except in cases of necessity; but experience seems to show that no ill effects are to be anticipated, and hence I have no hesitation in recommending it whenever there is the least anxiety as to autoextubation or subsequent adductor spasm. In my earlier cases, owing to unwarranted suspicion of the self-retaining tube, I believe I have taken great and improper risks in using the "special" tubes, although they are less unsightly and more ideal in effect. The perpetuation of the fistula by the "plug" also offers the distinct advantage of a sense of security in adults who are relieved of the dread of autoextubation, and in case of

clogging of the tube can remove the plug and cleanse the interior of the instrument by simply poking a feather through the opening. Indeed I was first forced to employ this kind of tube in Case XV.; the normal (metal) tube previously used became obstructed one day quite unexpectedly and so nearly produced asphyxia that a substitute arrangement seemed imperative. This woman thereafter cleansed her tube in the way suggested every morning and never again had difficulty.

After removal of any tube, particularly a large or special one which has been worn a long time, there is great danger of sudden adductor spasm and strangulation and an opening in the trachea already present is a great safeguard. The spasm, however, usually occurs some days or hours after the removal of the tube (and plug), and the "fistula," when not kept patent artificially, is apt to nearly or quite close before this dangerous incident. The usual curved cannula, which could be employed in the interim, is manifestly improper, as it drags the lower segment of trachea forward and pushes the upper back, and therefore I recommend a perfectly straight cannula so fitted to its shield that it can be pushed in or pulled out and thus stay with its inner extremity protruding into the lumen of the trachea. The diameter of the straight cannula should not be over one-eighth or one-quarter of an inch to avoid undue separation of the tracheal rings and approximation of their unsupported posterior extremities, and at the same time it must be large enough to permit, if need be, the insertion of a tracheal dilator through the fistula it maintains. This straight cannula only need be worn for the first two weeks after removal of the intubation tube. Subsequently to this period I have never met with an adductor spasm, and any other obstruction occurs slowly and can be met by reintubation.

The plugged tube cannot be made in small sizes, as either the plug or the tube or both must be constructed of vulcanized rubber, and there is a limit to the strength of this material. The plug and tube cannot both be made of metal, as the calcareous deposit which always takes place on the surface of a metal tube is very hard and firmly attached and might easily prevent the removal of the plug if the latter were likewise made of metal. The workmanship must also be of the best and very exact, because the least irregularity in correspondence between the lumen of the plug when screwed in place and that of the tube will cause rapid clogging. Possibly a metal tube with a hard-rubber plug is the best combination for cheapness, strength, and durability, and as the retaining rod and cap are also of metal it offers possibilities for attempts at electrolysis for those who believe in such treatment. The "clamped" tube is the only one I have found capable of construction in the small sizes for children under ten years, and it is very useful. The tube has to be grooved on its outer surface to smoothly and exactly

receive the clamp, as any irregularities or roughness will produce ulceration. The tube must be of rubber and the clamp preferably of silver, as a metal tube and a metal clamp would speedily become inseparable on account of the calcareous deposit previously mentioned. Also a clamp fashioned like an obstetric forceps is much better than one having the blades pinned together as in a pair of ice tongs. The disarticulated blades, though somewhat difficult to insert in a squirming, frightened child, will enter a very small hole and also there is no pin to break and drop into the trachea. The "cap" of metal then screws down snugly on the tube over the shanks of the clamp and should leave no rough or irregular interval to produce irritation.

The treatment of the adductor spasm, which is particularly to be dreaded after using large or "special" tubes for the cure of an hypertrophic laryngitis, has been partly outlined in the advice given as to maintaining the patency of a tracheal fistula. It is dangerous and must always be watched for after any prolonged intubation; as it is apparently due to an atrophy of disuse in the abductors a small tube which permits them to functionate and at the same time maintain respiration is a logical procedure. After determining, if necessary, with the aid of anæsthesia that the hypertrophic condition has subsided a normal tube of the size proper for the age and development of the child is inserted and left for about one or two weeks. During this period and for a week longer assistance must be constantly near at hand in case of autoextubation. If, however, a clamped tube has been used and the small straight cannula retained in the fistula the danger is very slight. The nurse or any attendant, should difficulty occur, can readily push the straight cannula or a curved one for that matter into the trachea. The straight cannula is apt to slip outside the cartilaginous rings, especially when worn with an intubation tube. A curved cannula, by the way, inserted alongside the tube or into a fenestrum in its anterior wall, I have found to be an utter failure.

A *cicatricial* stenosis, particularly if a hasty tracheotomy has at some earlier stage been performed, is often complicated by a contracted trachea with a spur opposite the fistula; and if there has been a tracheotomy for the autoextubation following laryngeal diphtheria, there is in addition probably more or less hypertrophic laryngitis. But even when there is a history or probability of considerable loss of cartilage (see Cases VII., IX., and XV.) and contraction of the larynx until the lumen seems practically obliterated (Case XV.), experience has shown me that the prognosis is still good. In the milder cases without a tracheal fistula insertion of a normal hard-rubber intubation tube for from one to six months will invariably effect a cure. Cocaine is a sufficient anæsthetic, and if the resistance to a tube of the proper size seems too great a smaller one can be employed for a few days and then a larger sub-

stituted. In the tighter and more deforming strictures general anæsthesia is required to permit the necessary preliminary dilatation. The bad strictures always have had a tracheotomy at some earlier period or need it urgently when first seen, and the fistula serves admirably for the introduction of urethral sounds which are excellently adapted for the purpose. Beginning then with the smallest size, which will pass through the contracted area, others are passed in rapid succession until the intubation tube can be inserted. Some judgment must of course be used to avoid the possibly fatal accident of making a laceration which may extend into the pharynx or œsophagus, but though I have done the operation many times and used very considerable force I have never encountered this or any ill results. Nevertheless the possibility of its happening must not be overlooked. Occasionally, as in Case XV., the stricture may be so tight or tortuous that its lumen may have to be opened by a careful dissection like a strictured urethra, but as long as any traces of mucous membrane are left and a tube can be passed from the pharynx into the trachea over or through the remains of the epithelium, there can be good hopes of a cure. Even very extensive loss of cartilage, as in Case IX., may be repaired, although Dr. O'Dwyer[1] cites one instance in which as the result of diphtheria the whole cricoid had sloughed out and this he considered irremediable except by tracheotomy. I should not, however, be inclined to condemn such a case without a trial.

In addition to the stricture, which is generally worse above the fistula, there will always be one at and just below this point, especially if the cannula has been worn long, and this also must be dilated like the one above. Then when inserting the tube the spur opposite such an old fistula in the posterior tracheal wall will be a serious obstacle to pass unless a small right-angled blunt retractor or hook, grooved on its convexity to receive and guide the entering tube downward and backward, is placed in the lower angle of the fistula, dragging the inferior tracheal segment forward. In adults I have several times had to exert all the force possible in two fingers pushing down from above on the head of the tube while counter-pressure was made externally on the larynx with the other hand to pass this point with its spur.

The comparative advantages of using a normal, a special, or a "clamped" or "plugged" tube in these cases have already been discussed. As a rule a combination of a plugged with the principle of a special tube is the best. The plug is a safeguard against clogging and autoextubation and maintains the fistula as a subsequent escape from the difficulty of adductor spasm; the feature which for brevity I have called "special" in the instrument means an enlargement of some portion of the diameter over the corresponding diame-

[1] American Surgery, May, 1900.

ter of the normal tube to exert a dilating pressure upon some particular part of the larynx or trachea. The normal tube is long, as it was designed by Dr. O'Dwyer to pass as deeply as possible into the trachea beyond any diphtheritic process. Its lower end is constricted from side to side to pass harmlessly through the glottis, the retaining swell is as large as will pass easily through the ring of the average cricoid, and from this point upward the tube is again flattened from side to side to fit the larynx. The "special" tube must always have the retaining swell, and this can be larger than the normal, as a diphtheritic swelling within the cricoid and consequent sloughing from traumatism at this point need not be considered; but at its neck or its lower end its diameter must be increased to stretch the stricture to be combated up to the largest possible normal lumen of the air passages. No exact measurement for this can be given, and they can only be approximately estimated and by a trial of different tubes. If, for example, the stricture is in the trachea at the lower end of the cannula (rare, though I have seen one case at the Willard Parker Hospital), the tube from the retaining swell down must be of the same diameter throughout and long enough to pass the contracted area. Again, if there is an hypertrophic laryngitis complicating a cicatrix the tube must have the diameter of its neck increased, although (unless it is a clamped or plugged tube) the transverse diameter of the retaining swell must always exceed that of the neck by three-thirty-seconds or preferably four-thirty-seconds of an inch.

A rough estimation of normal measurements which I have found useful is obtained by comparison with the patient's index finger, which is somewhere near the calibre of his trachea below the cricoid. The length of this finger is also about the distance from the suprasternal notch to the tracheal bifurcation.

The prognosis of a cicatricial stricture is difficult to give. Some cases seem to recover permanently after a few weeks or months of continued dilatation; others, especially those in which the rules for a proper tracheotomy have not been or could not be carried out, will relapse from time to time and prove very discouraging.

In a few instances I have attempted to dissect out all cicatricial tissue and, by accurate suture of the trachea and overlying soft parts over a well-fitting tube, to obtain primary union and so do away with these annoying and dangerous relapses. But the results have not been encouraging, possibly because asepsis is practically impossible. In Case XI., although the wound united throughout by first intention, the stricture was again evident within a few days after the removal of the tube. In the others infection took place and the wound broke down in whole or in part. On the other hand, persistence in the dilatation for months or years (Case VII.) has seemed finally to yield a permanent cure, and hence in general cicatricial strictures of the larynx or trachea should be continuously

maintained for at least six months. An attempt may then be made to do without the tube (with proper regard to adductor spasm), and at the end of a week the larynx and trachea tested for recontraction by insertion of the tube last worn. If none is apparent the test should be repeated once every one or two weeks for a month or more. At the first sign of recontraction the tube should be inserted and worn for another six months and then again tested.

But if the stricture is once allowed to contract seriously beware of attempting to intubate without preparations for tracheotomy. Such a recurrent stricture, especially if near a previous tracheal fistula, is apt to be very tight before it causes distress and very resistant to tubage. It is wisest to first attempt a passage with the Schroetter dilators, and if these fail do another careful tracheotomy in the old cicatrix when present and then dilate again with urethral sounds followed by intubation, as at first. Perhaps the safest rule after a cure seems obtained in these cases is intubation for a week every couple of months or oftener if required for several years or until no tendency to contraction can be detected during a considerable period. A pure hypertrophic laryngitis is, if anything, more difficult to handle than the cicatricial form of obstruction, as there is no time to think in the interval of changing the tube—generally for weeks or months the obstruction remains complete and the clean tube has to be immediately reinserted and it takes longer to cure the condition. But after it is once well I have never met with any recurrences of the stenosis. The average duration of treatment has been from about six months to a year, although one case recovered permanently within four weeks and another, complicated by numerous cicatrices to be sure, required nearly three years before natural breathing was finally obtained. This very long period was, however, largely due to my inexperience at that time.

Granulations as a cause of chronic obstruction are, I am convinced, rare, and when present are fortunately easily handled. Their removal, like any other new-growth, by cauterization, excision, or curettage is of course the first step and this can generally be accomplished through the mouth or tracheal fistula, although the latter may have to be enlarged to provide access. To prevent recurrence sources of irritation must be eradicated by making a new fistula or by changing the shape or length of the tube.

Granulations are most common at the base of the epiglottis around the head of the intubation tube, and Dr. O'Dwyer describes two cases in which they were finally overcome in this situation by so building up and remodelling the head of the instrument as to distribute the pressure it exerted over a larger area. In Case IX. the difficulty was satisfactorily met by using a smaller than normal tube. The principle of course is to remove the traumatism, or at least minimize it, and thus the details for individual instances can be readily adjusted.

A word as to feeding may be necessary. For the first few days after intubation liquids only are permissible and they should be taken in the Casselberry position with the mouth lower than the pharynx and it lower than the œsophagus. But it is surprising how quickly these patients learn to swallow a varied diet naturally and how few abnormalities they display. Even if an intubation tube should have to be worn permanently this is far preferable to the alternative of a tracheal cannula.

EMPYEMA OF THE FRONTAL SINUS.[1]

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The case I report illustrates so well the so-called closed empyema of the frontal sinus and certain peculiar phases of the disease that I have thought it might prove of interest. By closed empyema I mean that at no time could a discharge of pus into the nose through the frontonasal canal be detected. Though several careful examinations were made from time to time, this most important symptom of disease of the accessory cavities of the nose did not appear. The history of the case is as follows: The patient is now thirty years of age. He had most of the diseases incident to childhood, and since then has been healthy until the beginning of his present illness. His parents are living at advanced ages, and are healthy. In January, 1904, he had what he considered a mild case of "grip." He felt weak and languid and was forced to lounge about his room for two or three days. After this indisposition he convalesced rapidly, and was soon able to resume business. About two weeks later he was taken with a severe pain over the left eye. The pain recurred regularly every other day, and was peculiar in that it would begin at twelve o'clock noon, or between the hours of 12 and 4 P.M. If it did not appear before five o'clock in the afternoon the patient had nothing to fear until the next day. Occasionally the pain would miss the alternate day, but was sure to come the third day. On Sunday, if the patient remained perfectly quiet, the attack would be postponed until Monday, but if he took a walk or exercised in any way he was sure to suffer. The duration of the pain was always five or six hours. In the beginning of the trouble he suffered at times—usually in the morning—from dizziness or, to use his own words, he felt as if he were under the influence of liquor. The attacks always began in the left temporal region, with a feeling of numbness, and gradually extended toward the middle

[1] Read before the Medical and Chirurgical Faculty of Maryland, April 26, 1905.

line of the head with the point of worst suffering in the region of the supraorbital notch. The pain, at first slight, would grow more and more intense until, at the height of the paroxysm, the patient would walk the floor in agony. From this period of culmination, there would be a subsidence of the trouble, until it would disappear entirely. The patient consulted his family physician, who gave him anodynes, with no benefit. He then fell into the hands of two homœopathic physicians, neither of whom gave him any relief or an explanation of his pain. He then drifted around from one physician to another, and was in turn treated for stomach trouble, liver trouble, and nerve exhaustion. Thinking there might be some connection between the pain and his eyes, he saw an oculist, who told him that his glasses were correct. Two rhinologists were seen. One of them assured him that the pain had no connection with his nose and gave him such anodynes as he had not already taken. Finally, after several weeks of treatment, he suggested that the supraorbital nerve be cut. Just before consulting me the patient saw a prominent physician, who advised the injection of osmic acid into the nerve and the application of electricity over the affected area before having the nerve cut. He consulted me November 29, 1904. After hearing his history I felt that where so many had been unsuccessful I was doomed to failure also. From the history of the pain having followed "grip," from the periodicity, and from the fact that it was always localized over the left eye, I immediately thought of probable frontal sinus disease. I questioned him carefully as to a discharge into the nose or into the nasopharynx with a negative result. He denied having had any such symptom. Inquiries as to the condition of his stomach, liver, intestines, etc., revealed nothing worthy of note. His general health was good. He was wearing compound, hyperopic lenses, given him two years before by a very competent oculist, which corrected his astigmatism entirely. The edges of the lids showed a slight blepharitis. Inspection of the face gave no evidence of swelling in the affected area. Examination of the nasal fossæ showed on the right side a septal spur which did not interfere with respiration. In the left side, with the exception of an abnormal dryness of the mucous membrane, which I have several times noted in disease of the accessory cavities, there was nothing to throw any light on the mystery. After cocainizing, the most careful inspection failed to show pus anywhere in the nasal fossa. On being asked to localize the area of pain, the patient would place his finger in the middle line of the head just above the root of the nose and describe a semicircle outward almost to the external angle of the orbit. The pain never exceeded this area. Baffled by my examination of the nostrils, with the exception of the dry mucous membrane which added one link in the chain of evidence, I turned my attention to the supraorbital region. Transillumination of the two sinuses gave no information, one side being

as dark as the other, which was due, as I afterward discovered, to the thickness of the anterior wall of the cavities. Palpation and percussion over the left eye caused decided tenderness as contrasted with the opposite side. I then made a diagnosis of frontal sinus disease from the following points: 1. The pain followed what was undoubtedly an infectious disease. 2. The periodicity of the attacks quite common in frontal sinus disease. 3. The dryness of the nasal mucous membrane. 4. The extreme tenderness of the affected as contrasted with the sound side. The most valuable symptom was tenderness on percussion, which is almost pathognomonic of frontal sinus disease, while the group of symptoms seemed to point unerringly to the cavity as the cause of the headaches. Since he had had the pain for nearly a year, I advised an external or radical operation. The possibility of frontal sinus disease had not been suggested to the patient, and, not wishing to run the risk of possible deformity from an operation, unless it should be absolutely necessary, he decided to try the osmic acid treatment. The injections into the nerve were made, but did no permanent good. January 2, 1905, the patient came to my office in great pain and begged me to give him something to relieve him. He told me that he could see no relief in sight, and that he was fast becoming desperate. I prescribed for him, and again advised opening the sinus. He had suffered so long he was now ready to submit to any treatment that offered even a possibility of relief. On the afternoon of January 4th I operated at the Presbyterian Hospital, with the assistance of Drs. Carroll and Bray. Under ether anæsthesia an incision was made through the skin along the orbital margin, and the anterior bony wall chiselled through. This was perhaps three-quarters of an inch thick. On opening into the sinus there was a gush of pus. The mucous membrane was degenerated and covered with granulations. This was carefully removed with the curette, and the bone examined for a possible necrosis. The sinus was large, extending outward almost to the angle of the orbit and upward for at least an inch. After satisfying myself that no necrosis had occurred, I swabbed the cavity with chloride of zinc solution (20 per cent.), followed by alcohol and packed with iodoform gauze. The headaches have disappeared, and have not returned. The cavity is gradually filling up, which is the ideal result of the operation. Pus from the cavity showed staphylococcus pyogenes aureus in pure culture. The chief danger in these cases is the liability of necrosis, with consequent discharge of pus into the orbit and injury to the eyeball, or into the meninges, with the lighting up of a fatal meningitis. Logan Turner has called attention to the possibility of infection of the meninges through minute bloodvessels in the bone without any apparent necrosis.

The cardinal symptom of accessory cavity disease is the presence of pus in the nose. Its absence renders the diagnosis much

more difficult. M. Hajek, of Vienna, perhaps the greatest living authority on these diseases, says in his book (*Die Nebenhöhlen der Nase*, 1899) that in most cases of inflammatory frontal sinus disease pus is present in the nose. Exceptions to this rule are those rare cases in which the opening of the frontonasal canal into the sinus has a valve of mucous membrane, or there exists in the course of the canal an impermeable stricture. Such cases are doubtless exceedingly rare, and since there is a damming back of the secretion in the sinus, with eventual breaking through into the orbit and displacement of the eyeball, they do not fall into the hands of the rhinologist. The occurrence of such cases has been denied by Grünwald and Lichtwitz. Hajek himself has never seen such a case. And again he says that often the attention of the physician is attracted to the possibility of a frontal sinus disease on account of the intermittent, neuralgic character of a frontal sinus pain. He has often heard that a headache which comes on in attacks at the same time of the day is a typical neuralgia of the supraorbital nerve. Behind this typical, intermittent neuralgia how often has he found empyema of the frontal sinus. Particularly do the so-called influenza neuralgias of the trigeminus reveal themselves regularly as typical empyemata of the accessory cavities, especially of the frontal sinus. How easy it would be to heal many of these empyemata if the physician would only think of the possibility of frontal sinus disease and refer the patient to the rhinologist for immediate treatment, instead of after months of delay. Such words from Hajek must make an impression upon all of us.

It may be interesting to refer briefly to the pain of frontal sinus disease. It will be found in some cases that the patient will complain of pain only on alternate days. Such cases will simulate malaria, but treatment with quinine does no good. The pain usually comes on in the morning before eleven o'clock. The explanation is that during sleep there is no drainage through the frontonasal canal, and the sinus fills with pus. On arising in the morning there is a great pressure brought to bear on the opening of the canal, and this pressure produces pain until free drainage is established. I have never before seen a case where the pain came on in the middle of the day or in the afternoon. Such cases are certainly very unusual. In my case it is a question as to what became of the pus that was constantly being formed in the sinus. From the history of frontal sinus disease there should have been a profuse discharge of pus into the nose every other day. But repeated examinations, and the positive statements of the patient to the contrary, are certainly puzzling as to the escape of the pus. It does not seem possible that all, or even a large part of the pus, could have been absorbed into the system without causing decided symptoms, which the patient at no time had. I have not found an explanation of the afternoon attacks in frontal sinus disease.

I believe that many obscure headaches have their origin in some one or more of the accessory cavities of the nose, and that relief can only be obtained by intelligent examination and treatment of the same. The above case is presented as an illustration of a headache which could not possibly have been cured without an operation.

PERSONAL EXPERIENCES WITH EMPYEMATA OF THE FRONTAL SINUS.[1]

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At the present moment the therapy of frontal sinus affections is one of the much discussed and still unsettled problems in rhinology. During the last ten years various methods have been proposed, enthusiastically commended, and then allowed to drop more or less into disuse. These have varied from the simplest to the extremest methods of operation within the nasal canal, and from simple opening and prompt closure externally to the present much-advocated, very radical procedure of Professor Killian. As a contribution to the general subject the writer begs leave to offer for the consideration of this Association his own personal experiences in empyemata of the frontal sinus, together with some deductions from these experiences, and some observations as to what he regards as the probable future trend of operative procedure in this locality.

While the number of cases presented is not large, and while it is unsafe to reason too much from too limited experience, it is hoped that the deductions may have some value. The cases are reported somewhat in detail, the exact operative procedure being described rather than named after anybody in particular.

In his address at Atlantic City before the American Medical Association, 1904, Professor Logan Turner stated that what was really needed at the present time was an accurate report of the chronicity of the cases, the number of cases affected, the surgical method employed in operating, the treatment of the wound, and the results obtained as regards cure, relapse, the amount of disfigurement produced, and the fatal issue. As a contribution to such a qualified investigation I offer the present cases.

At the outset a certain amount of differentiation of cases must be made. I discard entirely that large group of acute inflammations of the frontal sinus which occur perhaps more and more often as accompaniments or sequelæ of grip, which may or may not be

[1] Presented as candidate's thesis, American Laryngological Association, Atlantic City meeting, 1905.

accompanied with purulent exudate, and which recover promptly under simple nasal treatment, sometimes with, sometimes without, removal of the anterior end of the middle turbinate.

The chronic cases of empyemata may also be divided into two classes: first, those in which the reasonable persistence of intranasal therapeutic measures has brought about success; and, second, those which, after the persistence for a reasonable time of such intranasal therapeutic measures as we have at our command, are still uncured or not sufficiently improved, and in which an external operation seems imperatively demanded.

Of the first group of cases the following four are examples:

CASE I. *Chronic inflammation of the left frontal sinus.*—Retired sea captain, aged seventy years. This case was referred to me on account of a great deal of mucopurulent discharge of long standing from the left nostril in a man of far from robust health and subject to attacks of heart difficulty.

There were numerous polypi in the left nostril, which were removed, leaving behind a steady discharge of foul pus in the middle meatus, with bare bone in the region of the frontal sinus outlet. Exploratory incision was made into the antrum, but there was no pus. I was able to pass a cannula directly into the frontal sinus after cutting away some diseased tissue in the region of the outlet with the Grünwald cutting forceps. The middle turbinate as a whole was not removed. The quantity of pus diminished, the odor gradually grew less, and the patient's general condition better. The sinus was syringed out every other day with 1: 8000 corrosive sublimate. On one occasion this syringing made him dizzy, requiring an hypodermic of strychnine and morphine before he was able to go home.

The discharge steadily diminished, and on April 1, 1902, he having come under observation on November 26, 1901, there was absolutely no pus discharge and he felt perfectly well. He was seen occasionally from this time until December 10, 1903, and at no time was there any sign of discharge from the nose or any trouble in the sinus.

This case shows what can be done under apparently difficult conditions even with a foul purulent discharge. I was not allowed from start to finish to do anything but the mildest kind of operations such as could be done with a snare or a few strokes of a cutting forceps, yet, to all intents and purposes, this patient was as perfectly cured by treatment within the nose as he would have been by any external operation. Three years have now elapsed without any return of the trouble. I have recently seen him and he remains in good health. There has been no nasal discharge since the cessation of treatment.

CASE II. *Subacute inflammation of the right frontal sinus.*—Mrs. W. H., aged twenty-six years. First seen January 28, 1903. Had

history of pain in the head following grip three months ago, with a discharge from the right nostril for five weeks. Complained of pain in the right side of the head and soreness above the inner angle of the eye. Pain was very much worse when leaning over and of a throbbing character. There was pus in the right nostril. The middle turbinate was crowded against the septum and against the bulla so that the space for drainage was very poor. Could not probe the sinus. Provisional diagnosis of subacute empyema of the right frontal sinus.

The next day removed anterior portion of the right middle turbinate in order to get better drainage. The pain over the right eye soon diminished, and in a few days she was quite comfortable. The pus discharge continued, gradually diminishing. I was able to carry the cannula up into the sinus and syringe it out, but only with the greatest difficulty.

Two months later there was still discharge from the nose in spite of daily treatment. It was difficult to wash out the sinus very successfully, and I thought the only cure would be by means of an external operation, which I advised but which was refused. A few days after this I removed the rest of the middle turbinate and was able to get farther up toward the opening to the frontal sinus, but its washing out was only fairly successful, owing to a very narrow passage.

As, however, the patient refused any external operation and the discharge slightly lessened, and as the infundibular opening was gradually enlarged so that various solutions could be carried up into the sinus, the idea of an external operation was given up, and on May 19th there was apparently a cure of the sinus condition and the case was dismissed. Six months later there was no apparent difference on transillumination between the two sinuses, and no sign of trouble in the nose. At the present time, a year and a half later, there has been no further trouble. Her husband, however, reported to me that following an acute cold this winter there was a little sinus pain, which soon disappeared.

In this particular instance, although there has been an apparent cure by intranasal methods, I think it would have been better surgery to have opened externally, as I think there is a likelihood of a return of the trouble after an attack of grip or any acute infective disorder.

CASE III. *Chronic inflammation of the right frontal sinus, with acute exacerbation.*—Dr. E. E. R., aged forty-five years. Has a history of several times in the past having what was called severe neuralgic pains of the right side of the head, right eye, and region of the right sinus. At one time gave up his work for nearly an entire year, not having any relief at all until after three or four months in the summer in the dry air of the highlands of New Hampshire, after which he was free from this pain for several years. While travelling in a

sleeping car recently, March 12, 1904, was taken with severe pain in the region of the right frontal sinus. Had no relief from treatment. Had to give up his work, that of an active physician.

Examination of the nose showed the right middle turbinate engorged, so much so that adrenalin did not blanch it. No pus was visible. The right middle turbinate was at once removed, giving some relief to the severe pain. Could enter the sinus with a probe. The pain greatly diminished until, four days after seeing him, was able to wash out the right frontal sinus through a cannula, removing a considerable quantity of mucopus, with immediate relief of all the symptoms. Washing was continued, and at the end of a week he was well and has continued so to the present time.

This was one of those cases of severe involvement of the frontal sinus in which, if the removal of the middle turbinate can be promptly done, the sinus drained, and relief of the engorgement brought about, a cure will be effected without further operative procedure.

CASE IV. *Subacute inflammation of right frontal sinus.*—A. S., aged forty-four years, designer, came on April 12, 1904, complaining of great pain over the right eye, which he thought was due to trouble with his glasses. The oculist reported that they were all right. All nasal structures on that side congested. Treatment, nasal irrigation.

April 14th. Pain has been almost continuous. Unable to sleep. I at once removed the middle turbinate on the right side. After the operation was able to pass a curved cannula directly into the right frontal sinus, washing out some thick purulent debris.

15th. In spite of the pain which followed the operation and the necessity of wearing a cotton tampon, has had a better night than for weeks. Washed out the frontal again. April 16th, washed out the frontal sinus. No further pain over the eye.

19th. Tried to go to his work, but gave it up and went to bed. Some reaction evidently following the operation. Washed out the sinus, but no pus. From this time on the nasal discharge entirely ceased. He had an attack of acute rheumatism, for which he went into the hospital, but there was no pain in the region of the frontal sinus, and when seen the last time, on May 12th, there was no discharge and he was perfectly well.

This was a case of subacute inflammation of the frontal sinus with an acute exacerbation in which the prompt establishment of free drainage cured the case. It is also one of those cases which, improperly diagnosed, tends to go on to a chronic condition with constant pus discharge.

The following group of cases belongs to Class II.—viz., those which remain uncured or insufficiently improved after faithful use of intranasal measures, or in which, for urgent or other reasons, intranasal measures are manifestly not indicated.

CASE V.—Mrs. G., aged thirty-five years, seen October 17, 1899, on account of severe headaches. Examination showed pus in each nostril with polypi. The polypi were removed and then each middle turbinate.

December 15, 1899, the right frontal was opened externally at the inner angle of the eye underneath the supraorbital ridge. There was fluid pus but no granulations. The opening was extended down into the nose; the external wound was treated as an open wound for two weeks and then allowed to close, at which time the probe passed easily into the nose from the right sinus. There was improvement of the pain but the pus discharge continued, and later complaint was made of pain over the left sinus. This was then opened in the same manner and the right one reopened. The two did not communicate. The opening into each nose was made larger with good-sized curettes; iodoform gauze wicks passed through, followed in a few days by good-sized rubber drainage tubes passed clear into the nose and retained for four weeks. The wound was then allowed to close externally. The patient's general condition was much improved, although some pus discharge continued, varying more or less in amount.

A few months later the sinuses were again operated on and larger drainage tubes kept in for several weeks. There was improvement as before, but the pain returned again some months later and the discharge, which had never stopped, became worse, so another operation was done July 27, 1900. This time the incisions were made above the supraorbital ridge. It was found that the right sinus, except at its most dependent portion, was completely closed with osseous growth as the result of the former operation, but there was pus in the left sinus.

For something over a year the condition was not very troublesome, but November 8, 1901, on account of throbbing pain and some pus discharge, the sinuses were each opened at the most dependent portion of the inner angle of each eye, found nearly closed with bone, the trouble being mostly at this time in the cells in the dependent portion of the sinus. All of the ethmoidal cells which could be reached were curetted and destroyed. The rubber tubes were inserted and retained five days, followed by silver tubes worn for three weeks. She was seen occasionally for some months, then I observed that there was an empyema of the left antrum. This was due to infection, as on previous occasions both antra had been found free from pus.

On July 23, 1902, a radical operation for the empyema of the antrum was performed through the canine fossa, followed by rapid healing; the antrum at the time of the operation containing considerable granular debris. After this was done she was not seen for a year, during which time she became pregnant and had a normal labor. At this time nothing special was found

in the nose, although she still claimed to have more or less pus discharge.

On July 12, 1904, she was examined. There was a little mucopus in the nose, but she was practically well and in good health, all the pain having disappeared.

On April 11, 1905, re-examined; both frontal sinuses apparently obliterated. There was a little dried scabbing in the floor of the nose, due apparently to atrophic conditions. Probe detected bare bone in the right sphenoid; left sphenoid could not find the opening. The patient's general condition good. This case is a cure so far as the empyemata of the frontal sinuses are concerned. It is doubtful whether all of the ethmoidal cells are in a healthy condition.

Criticism. This was a case of extensive ethmoiditis with involvement of each sinus, and later infection of the left antrum from the sinus. My early operations were without doubt incomplete. Not having been followed by complete obliteration of the sinuses, there was continual reinfection until, as a result of the many operations, a final cure was effected. The early operations were too timid and the openings into the nose were not large enough nor the infected ethmoidal cells thoroughly destroyed. The sinuses were not extensive in area, and had complete obliteration, by packing, been attempted, a cure would no doubt have been effected much earlier.

CASE VI.—Mr. F. W. N., aged thirty-two years, milkman, resident of Martha's Vineyard. Seen in November and December, 1898, for polypi and pus discharge lasting a long time. The polypi were removed with so much improvement that he did not present himself again for a year, at which time the pus was still present and the polypi had recurred.

The middle turbinate was removed, and under ether January 12, 1900, I made an opening into the right frontal sinus at the inner angle of the eye, making an incision in the ridge from the supraorbital foramen to the nasal process of the superior maxilla. The opening into the sinus was made at about the mediate point of this incision, around the inner angle of the eye. The sinus was filled with free-bleeding granulation tissue, all of which was curetted out, and the opening enlarged so as to get free drainage into the nose. A wick of iodoform gauze was carried down into the nose, followed with rubber drainage tubes, and at the end of two weeks, the sinus being apparently in good condition, the external opening was allowed to close, and the patient returned home.

He was seen once or twice afterward and then not again for something over a year, when he reported that there was some pus in the right antrum, easily blown out when the right cheek was uppermost. He was not seen again for a year when, January 7, 1902, he reported that there was some discharge from the nose in winter. Examination showed no pus presenting at the frontonasal

canal, but a little bare bone could be felt with the probe, and there was no doubt but that the sinus was not perfectly healed.

April 10, 1905. This patient has just been re-examined. There is no sign whatever of pus in the nose, and he states that there has been no discharge from the frontal sinus so far as he knows for five years. Two or three times in the winter he has had a little sense of pain in the right maxillary sinus, followed by a discharge of mucus and mucopus, quickly stopping. This would apparently be due to a mild attack of inflammation of the maxillary sinus to which reference has already been made as having occurred in the past. Transillumination of both frontals was done, the right frontal showing almost entire obliteration, the left frontal extending high up on to the forehead, with two or three septa plainly visible. Transillumination of the two antra showed each one perfectly free, so that at the present time a perfect cure of all the accessory sinus trouble has apparently occurred.

Criticism. This was another one of the cases in which, viewed in the light of later events, I think that complete obliteration of the sinus should have been attempted at the original operation, and that presumptively the small amount of pus remaining, with the possibility of colds and inflammatory action in the nose, very likely infected the maxillary sinus. As, however, the patient was dependent upon his daily labor for existence, and as he lived where he was practically inaccessible, it seemed as though the less radical operation would probably answer, and this has in fact occurred, as with the lapse of time a perfect cure has resulted.

Case VII.—L. J., student, first seen April 5, 1899, when sixteen years of age. Brought on account of fear that he had tuberculosis. Had had a cough for ten years. One sister had died of tuberculosis and he had spent the previous two years in Colorado and New Mexico. Was unable to find any tubercle bacilli in the lungs, but micrococcus tetragenus aureus and streptococci were abundant. Under treatment the chest conditions improved. During this time he had an acute otitis media in both ears, relieved by incision. Was under my care at intervals for a year, during which time I removed a few polypi from the right nostril, and a year later, June 27, 1900, recorded that the nose was running a good deal. There was some discharge apparently from the left frontal sinus. Transillumination did not show any very well-defined shadow. He was relieved by washing out the nose with hot Seiler's solution, which treatment he continued on his own account. About this time he entered Brown University, completing his course there in about three years.

From June 27, 1900, to May 29, 1903, I have no record in regard to him, though I saw him occasionally. The lung conditions had very much improved. At this time he came complaining of discharge from the nose, which he said had continued more or less for

several years. Examination showed pus in the nostril, and exploratory puncture demonstrated it in each maxillary antrum. These were washed out at intervals and numerous polypi removed. The right middle turbinate was also removed. Diagnosis of empyema of frontal as well as maxillary sinuses, and operation advised.

On June 17, 1903, he was operated on in the hospital, the left sinus, the left antrum, the right sinus, and the right antrum all opened. Pus and diseased mucous membrane and polypoid degenerative material was found in each cavity, the antra being practically filled. The frontal sinuses were opened at the inner angle of the eye, underneath the supraorbital ridge, the greater part of the front wall of each being cut away, while the frontonasal canals were each enlarged sufficiently to take a large drainage tube, and the adjacent ethmoid cells curetted. The entire mucous membrane of each sinus was curetted out. The sinuses did not communicate and were of ordinary size both as regards extension upward and outward. A large drainage tube was carried down into the nose from each sinus. Each maxillary antrum was opened through the canine fossa; all the diseased mucous membrane curetted away, and a large counter opening made underneath the floor of the nose. All the cavities were packed with gauze.

The after-treatment was uneventful so far as the maxillary sinuses were concerned. The gauze was kept in fifteen days and then not replaced. A gutta-percha obturator was kept in each antral opening for about two weeks afterward. Then they were allowed to close. There has been no further trouble from them. The opening underneath the inferior turbinate has continued to the present time.

The frontal sinuses at this time were granulating in a healthy way and were being allowed to close in, gauze dressings being retained, the tubes having been removed about ten days after the operation. The gauze put in was saturated with balsam of Peru. At this time I was taken ill with typhoid fever and did not see the case again for nearly three months, during which time he was under the care of a colleague, who allowed the openings to close up. When I saw the patient they had broken out again at the surface, and the right one, he said, had never entirely healed. Both were curetted. The left one had a very little superficial pus. The right one showed some soft bone under the supraorbital ridge and some thick pus with some polypoid tissue at the nasal outlet. Gauze drains were replaced.

October 24th, three weeks after, both sides were closed; the right nostril was discharging a little pus, the left none. November 18th, a month later, there was a little accumulation of pus from the right sinus. December 24th, the left middle turbinal, which had not been previously removed, was taken out and a polypus from the region of the right frontonasal canal.

February 2, 1904, the right sinus broke out again with slight pus discharge, but the probe followed only a narrow track. Did not find much of a cavity. Opening into the nose still patulous. Curetted.

May 9, 1904, reported that he had some mucus discharge from the nose, but did not find any source from which it came. May 28th found several small polypi in the right infundibulum and just a suggestion of pus. Curetted a little. Some anterior ethmoidal cells not wholly cured.

July 15, 1904, pocketing of pus had again occurred on the right side. On opening was able to pass a probe through a small opening down into the nose. The left sinus remained completely closed. Remnants of the right sinus were very small. Curetted thoroughly. Washed down into the nose with pyoktanin solution, and allowed to close again in the hope that complete obliteration would this time take place.

At present, September, 1905, the sinuses are apparently well, but there is occasionally a slight mucoid discharge from the ethmoid region, for which I have recently removed several of the anterior ethmoid cells, and regard him as practically well. No appreciable scar remains as the result of the operative measures.

Criticism. As the sinuses in this case did not extend upward or outward beyond the ordinary average limits, and as the external wound could have been kept open sufficiently long for the obliteration method, I think this case would have done better if, at the time of the original operation, the opening down into the nose had been made larger, the anterior ethmoidal cells curetted more vigorously, the bone opening continued lower down, cutting away a portion of the nasal process of the superior maxillary, and then the external wound maintained by gauze packing until complete obliteration had taken place. This was in my mind to do, but unfortunately my own illness prevented, nor had I at that time become so thoroughly convinced as at present of the absolute necessity of obliterating the sinus in order to prevent a recurrence. Nevertheless I believe that a cure has finally occurred.

Case VIII.—G. F., aged twenty-six years. First seen June 21, 1899, having developed a frontal sinus trouble as the result of trauma, he having been injured in a railroad accident. The exact nature of the trouble was not discovered for some time, the case having first been reported to me as a case of atrophic rhinitis with a foul discharge. Was treated by the usual washing methods for a while, with opening through the canine fossa made with motor drill. The pus continuing in spite of pretty free drainage from the antrum, the question of frontal sinus trouble came up. He was seen by Dr. Leland, who thought the frontal sinus involved and advised an operation on the sinus. He was complaining a good deal of headache, which seemed to be due to the frontal sinus trouble.

On June 19, 1900, the right frontal sinus was opened at the inner angle of the eye. Pus was present but not in large amount. There was no odor and very little granulation tissue. Sinus area extended up about one-half inch and outward one and one-half to one and three-quarters inches from this point. The opening down into the nose was enlarged and a large-sized piece of gauze wicking put in. The right maxillary antrum was then freely opened through the canine fossa above the place previously opened and filled with gauze. The frontal sinus opening was packed with gauze two or three weeks, the gauze being followed by a small gold tube, which was worn for two or three months. Its removal was followed by complete closing of the frontal sinus opening, the sinus being completely obliterated.

This was in July, 1900, and there has never been any reopening of the frontal sinus, nearly five years having now elapsed. There has been occasionally a little bit of mucopus in the antrum, and he has insisted all these years on maintaining a small opening from the mouth into the antrum, which he washes out once or twice a week. I have not been able for some three years to demonstrate any pus in the antrum, but he feels better if he can wash out the antrum occasionally, and now and then imagines that he has a little pus. Practically, however, the case has remained perfectly well for the last four years, and the frontal sinus has never broken out.

In this instance, with a comparatively small sinus and rapid formation of granulation tissue after the operation the gold tube seems to have worked as well in maintaining the opening and to have been rather more comfortable to wear than the gauze. One reason for the good result may have been due to the fact that infection was secondary to antrum trouble and there was not extensive involvement of neighboring ethmoidal cells.

At the present time there is no appreciable scar, close search being required to determine the site of the operation.

Case IX.—O. M., aged seventeen years, spinner. First seen February 11, 1902, at which time she complained of a nasal discharge and pain in the region of the right frontal sinus. Gave a history of the discharge from the right side of the nose for one year with pain in the right side over the right eye. Examination showed pus in the right nostril, coming from high up in the nose. Probe detected bare bone in the infundibular region. Transillumination was negative. Probable diagnosis of empyema of the frontal sinus made.

February 15th removed the entire middle turbinate on the right side. Diagnosis then positive. Could pass the cannula into the sinus and syringe out the pus.

On March 5th was operated on at hospital for empyema. Incision made beneath the supraorbital ridge at the inner angle of the eye. Periosteum laid bare. Bone darker than normal; thin over the

sinus wall; opened; pus and granulations; no odor. Enlarged the opening; removed the granulations. Enlarged the opening into the nose so that a fair-sized rubber drainage tube was easily passed into the nose. There were no symptoms pointing to antral trouble, so the antrum was not opened. There was no connection between the two sinuses. The area of the frontal sinus was not excessive, not going very high above or outwardly.

Treatment in the hospital uneventful. Occasional slight temperature rise for a few days. Dressing changed on the third day for the first time. New tube put in on the seventh day. Left the hospital on the twelfth day, at which time the drainage tube was still in and the pus discharge very slight. The tube was removed a few days later, followed by gauze in the external opening. Pus discharge almost stopped, but not entirely. From this time until May 3d the nose and the external wound were washed out every other day, the external wound being practically closed in.

As there was still some pus from the nose, I punctured the right antrum and found pus, and on May 7th, two months after the first operation, again put her in the hospital and did a radical antrum operation. There was some pus but not much degeneration of the mucous membrane. A large opening was made into the canine fossa with a counteropening underneath the anterior end of the inferior turbinate. This was packed with gauze. The frontal sinus was again curetted. At the external wound there was no pus, but there was more or less rather poor granulation tissue. The opening was packed with gauze. Slight rise of temperature after the operation, but hospital record practically negative.

On May 15th the communication between the sinus and the nose had closed. There was some pus discharge from the frontal sinus wound around the gauze, but it was granulating and filling in. May 20th there was no pus in the nose from the frontal sinus. On May 24th the frontal sinus wound was still discharging a little pus. She was given chloroform in the office, and some low form of granulation tissue recuretted, and instead of the gauze I put in a curved silver tube, carrying it down into the nose preparatory to letting the cavity close.

The antrum wound had had the gauze dressing continued and had done perfectly well. From May 27th to June 5th there was no apparent pus from either cavity, and the case, being apparently cured, was discharged.

On August 28th, three months later, she returned with the opening in the frontal sinus broken out, small accumulation of pus causing some œdema in this region. Under chloroform skin incision was made, the opening down into the nose found with the probe, and the cavity packed with gauze. The dressings were then done every other day, and on September 9th there was apparently complete healing, and the case was again dismissed.

Two months later she was again seen. There had been no trouble. Not seen again until ten months later, June 2, 1903, when she returned with a small area of pus presenting from the right sinus. Chloroform was given and the pus evacuated. It seemed to be in the surface only. The sinus was apparently obliterated and the probe did not go any distance. A gauze drain was put in, and on June 9th there was apparently complete healing. Case has been well ever since and there is no deformity.

Criticism. In this case the antrum was probably infected from the frontal sinus, and the diagnosis of the antrum trouble should have been made coincidently with the diagnosis of the trouble with the frontal, and both operations should have been done the first time providing there had been pus in the antrum.

With reference to the treatment of the frontal, in which there was a recurrence of trouble in the granulating area on three different times, this should have been treated by the method of absolute obliteration with gauze drainage from the start, and the opening into the nose should have been made sufficiently large so there would be no likelihood of that closing during the period of granulating above. So far as can be seen, a perfect cure resulted with complete obliteration of the sinus, but the time required seems to me to have been longer than it should have been.

Case X.—Mrs. E. S., aged forty-six years. Came on account of catarrh on April 10, 1903. Examination of the nose showed numerous small polypi around the anterior end of each middle turbinate. These were removed, and on April 15th the right inferior turbinate was removed. She suffered greatly from inability to sleep. April 25th several polypi had come down from the ethmoid region where the turbinate was snared off.

May 15th some pus from the frontal sinus. Washed out without difficulty. Probable diagnosis of old empyema of frontal. The sinus was washed out for a number of days and the infundibular region curetted of polypoid tissue. The pus lessened in amount but continued. The antrum was opened exploratorily but no pus found.

On June 17th the right sphenoid sinus was opened and cleaned out with a curette. The walls were rough, but no apparent pus. On June 24th the right frontal sinus was opened under ether. It was small in area with soft anterior walls, filled with pulpy mucous membrane, which was removed entire, the opening into the nose enlarged, large drainage tube inserted, and the sinus cavity packed with gauze.

The after history is uneventful with the exception that on July 7th, two weeks after the operation, she complained of seeing double. The affected eye could be rotated in every direction, and I was not conscious at the time of operation of injuring the pulley of the superior oblique. The double vision gradually lessened, and on

November 28th had entirely disappeared and she was perfectly well. This case has been recently seen and there has been no recurrence. The opposite side, which had shown some polypi, has ceased to trouble her, and there is no pus discharge or any trouble whatever.

Criticism. In this case the anterior bony wall of the antrum was so soft as to break away easily under the use of a curette, hence extraordinary care should have been taken when in the region of the pulley of the superior oblique. This is the only case of double vision which I have seen following frontal sinus trouble.

CASE XI.—W. G., aged twenty-nine years. First seen March 22, 1903, on account of pus in the right nose and right ear. Said to have followed an attack of grip. There was an otitis media purulenta with perforation.

The right nostril was filled with polypi and there was a constant pus discharge. Exploratory puncture into the antrum showed the presence of pus, and the case was treated by washing methods. The polypi were removed, after which it was possible to probe the frontal sinus and wash out through a cannula. The frontal sinus was irrigated daily with a great deal of care, and this method of treatment carried on an entire month without much if any improvement, except temporarily. Radical operation was then advised.

The right frontal sinus was opened underneath the supraorbital ridge on the right side. Pus immediately welled out in considerable quantity. The opening was enlarged. The sinus itself was very large, extending clear over to the left side as far as directly above the supraorbital foramen on the left side, and quite a distance up on the forehead. I thought at first there was a communication between the right and left sinuses, and was on the point of opening the left sinus but did not do so. Cleaned out considerable polypoid tissue in addition to the pus. Opening into the nose enlarged and drainage tube inserted.

A radical operation was then done on the antrum. It was found partly filled with large polypoid masses, some of them of considerable size. The usual counteropening was then made into the nose. He was kept in the hospital two or three weeks. Had more or less pain and swelling of the eye.

At this time I was away for three weeks, when he was under the care of another physician. Both cavities had been washed out daily. This was on May 16th. Examination showed the antrum apparently nearly healed. The frontal sinus had a silver tube in it. Opening down into the nose still present and of fair size. Examination showed a very large sinus going away over the left side.

Decided to open the left sinus for fear that the two sinuses communicated, and if this were not done some pus pocket might be left which would cause trouble in the final healing. On May 17th the left sinus was opened under ether. Could not find any communi-

cation whatever with the right one. This sinus was very small and quite limited in extent, communicating freely with the left nostril. The mucous membrane was pulpy, detached at one place, and on pulling came away entire. Made a good-sized opening into the nose. This sinus promptly healed by the obliteration method, and has caused no trouble since. The other sinus continued to discharge a little. The patient, however, went back to work. Felt perfectly well. Gold tube was kept at the external angle of the wound, and he was seen by me once or twice a week.

October 24th, had been at work through the summer, but reported at this time on account of some pain in the left eye. The opening between the two divisions of the sinus had closed, and pus had accumulated in the left side. This portion of the sinus was curetted as thoroughly as possible under ether, and gauze drain put in.

For the next two or three months there was slight discharge. Various things were used for syringing and the discharge became small in amount. Still there was always a little. During the following spring solutions of pyoktanin and boric acid were used to wash out the cavity, and under this the discharge became almost nothing. The opening into the nose continued, and on January 30th, at the meeting of the Eastern Section of the American Laryngological, Rhinological, and Otological Society, he was seen in consultation with Dr. C. G. Coakley, who thought that permanent cure would not be reached until every particle of mucous membrane was removed, which could be done best, in his opinion, by taking off the entire front plate of the sinus. It might be attained by opening up through the already obliterated left sinus. As, however, the patient was in good condition, able to labor, but unable to afford the time to give up work, it seemed to me doubtful if any further operation was then advisable.

From this time for the following four or five months the discharge was almost nil. Four, five, or six days would elapse without any, and then there would be a little at the angle of the eye. Examination with the probe on June 15th showed the sinus to be apparently closed on the left side, but there was still some bare bone to be filled in the region of the supraorbital foramen on the right, and the probe passed readily down into the nose. Curetted toward the outer portion of the cavity to the right and a drop or two of pus came out. Removed a couple of polypi from the nose. At this time I used the pyoktanin in powder form, blowing it into the cavity.

From July, 1904, to December, 1904, he was seen by me only three times. On December 14th he reported there had been no pus discharge for an entire week. The probe did not hit any bare bone. There was almost no trouble in the nose. Up to the present time an occasional polypus had formed at the nasal outlet of the frontal sinus opening.

There has been no discharge of any account since December. Recent examination (September, 1905) shows small external opening into right sinus, which is to be closed by suture. The portion of the sinus leading to the left side obliterated, or else, which is more probable, division wall grown up so there is no communication. Right sinus much smaller. Opening into the nose easily found with a probe. No discharge of any amount and no polypi.

Criticism. This is one of the cases where, in the light of subsequent history, either an entire resection of the front wall of the sinus ought to have been done at the time of the first operation, or else at the time the second operation of the left frontal sinus was done, this should have been opened clear up so as to throw the cavity of the right and left into one, thereby giving better access to the cavity of the left sinus. The entire treatment consisted of drainage only, and as I was unable to remove the entire mucous membrane at any one operation, the case can hardly be called an absolute cure, even though for the last year or year and a half there are practically no symptoms. Should he have an acute infection or an attack of grip or something of that sort, I think this sinus is liable to be reinfected, and although I have an apparent cure, I can hardly report this case as an absolute cure. At the same time the patient, who was very much run down physically when first seen, is strong and well and hearty, and has had no pain or discomfort from the sinus for more than a year.

Case XII.—Dr. C. First seen January 27, 1904, with a history of illness for several weeks and a left-side nasal discharge, for which his physician wished me consulted. Examination showed creamy pus, coming apparently from the left frontal sinus, perhaps from the antrum as well. Transillumination showed extreme darkness as compared with the opposite side. There was some darkening over the left antrum. Exploratory puncture, however, showed the antrum to be clear. Nose was washed out for several days, giving some relief, and February 8th he had an acute otitis media, for which the drum was incised and gauze drainage used. There was some mastoid tenderness, but recovery from the ear trouble occurred without real trouble with the mastoid.

The sinus was washed out with pyoktanin solution for one or two weeks. Soon after this pus appeared to come from the right frontal sinus as well as the left. Both middle turbinates were then removed. The discharge from the sinuses diminished but did not disappear.

April 15, 1904, decided to do a radical operation. Opened the left frontal sinus by curved incision between the root of the nose and the inner line of the eyebrow. There was no fluid pus but a pulpy condition of the entire mucous membrane. Sinus in area about the average, not running up on the forehead to any great distance, though farther on the right side than the left. The entire mucous membrane was pulpy, thickened, and everywhere diseased. Re-

moved it entire with thorough curetting. Underneath the membrane the bone seemed hard and sound. Just above the root of the nose was a small opening about 3 mm. in diameter communicating with the opposite sinus, and through which it is probable that the left sinus infected the right, although there seemed to be no direct communication between the two membranes. After thorough curetting of the interior, the probe was passed down into the nose and this opening enlarged to carry good-sized rubber drainage tube. It was fastened above and below and the cavity packed with gauze.

The right sinus was then opened in the same way, the front wall, however, was very much thinner on this side, and the periosteotome easily penetrated the sinus when engaged in stripping the periosteum. On incision through the mucous membrane of the sinus, fluid pus was found to fill the sinus. This sinus extended quite a way over the eye and also above much farther than on the left side. The entire mucous membrane was curetted out and removed, and the opening down into the nose enlarged as before and drainage tube inserted. The bony opening between the two sides was then enlarged and gauze placed between. Each cavity filled with small strips of iodoform gauze.

The after-history of this case is uneventful. It was kept packed with iodoform gauze, soaked at intervals in balsam of Peru, and changed every day or every other day, and on August 12, 1904, the case was dismissed, complete healing having taken place. The point which filled up last, and which was in the end the most annoying, was the small bony septum between the two sides, to which the granulations did not seem to attach very well, probably because of its insufficient blood supply, and I regretted that I had not enlarged the opening between the two sinuses more. This is the only criticism I have to offer on this case. Healing up to the present time has been complete, and the patient, who had suffered from various vague head symptoms for many years, is better than he ever remembers himself to have been before.

Case XIII.—M. B., aged twenty years. First seen with nasal symptoms on March 2, 1900, at which time some pus presented from underneath the left middle turbinate and two small polypi were removed from this space. Probe was carried up into the frontal sinus. Transillumination showed opaque left antral region as against lightness on the opposite side. Pus was very apparent in the middle meatus. Diagnosis made of pus in the antrum, perhaps in the frontal.

March 7th was operated on under ether, a radical antrum operation being done. The antrum contained pus and soft, pulpy mucous membrane. This was entirely removed and the usual counteropening made into the floor of the nose. Frontal sinus not opened. The healing of the antrum was uneventful, but after this occurred

there was some pus still apparent in the nose, which did not disappear after syringing, and on April 23, 1900, the frontal sinus was opened underneath the supraorbital ridge at the inner angle of the eye. The bone was very thick. Pus abundant and very foul. Rubber drainage tube carried down into the nose and maintained for some time.

On May 14th the cavity had been allowed to close, trusting to nasal drainage. On June 8, 1900, there was a drop or two of pus exuding through the wound, so that a small curette was put in. Cavity curetted and packed with gauze. Healing of the external wound promptly took place, and I did not see him for some time, but on August 7, 1900, he complained of pain. Could not find any sign of pus in the external wound or nose. Seven days later, however, there was some sign of pus externally. This was curetted. Gold tube placed in for drainage and the probe carried down into the nose, readily finding the remnant of the opening made for the tube. Tube was maintained as long as possible, but the latter part of August the opening had closed, and September 19th, the case showing a tendency to continually reopen, he was again etherized. The frontal sinus recuretted thoroughly. Some soft bone found. The opening into the nose reopened and enlarged and the whole packed with gauze. By October 25th the opening had nearly closed, gauze having been kept in continuously, and the opening was allowed to heal up.

On November 8, 1900, the wound broke out again. There was a small amount of pus present. The whole area of softened bone again curetted. Small piece of gauze inserted. November 17th and 22d drop or two of pus found in each instance, and the latter time was able to carry the probe clear down into the nose. Syringed some peroxide through. The opening again allowed to close.

December 3d pus again found in considerable amount in the upper portion of the frontal sinus. Curetted again. Piece of gauze put in. December 10, 1900, under cocaine, curetted the upper portion of the frontal sinus over against the dividing line from its fellow of the opposite side. Put in gauze drain again. Washed out every day for the next month. December 29th no pus found in the sinus. January 15, 1901, a drop or two of pus showed in the angle of the wound. Frontal was again opened with the knife. On February 11, 1901, there was apparently complete cure.

For the next three years there was no sign of pus or trouble or anything of the kind. The young man went to the College of Physicians and Surgeons, New York, to study medicine, where early in April, 1904, having had no trouble whatever for three years, he was taken with an attack of grip while on ambulance service; had pain and signs of pressure over the right eye in the region of the scar of the former operation. He was put in St. Luke's Hospital, New York. Incision made. Some pus found. This incision was

allowed to close, then reopened, and fistulous opening had been maintained for three weeks at the time I saw him.

This was on May 20, 1904, at which time examination showed a fistulous tract, very narrow at the soft parts but leading into a wider, but not very large, cavity in the bone, apparently not communicating with the nose, of limited area, and very painful to the touch. Managed to curette a little. Enlarged the opening sufficiently to get a tube 2½ mm. into the opening. Curetting of this area was very painful. It was evident that no healing was going to take place from the bottom until the external wound was enlarged and the drainage improved.

He was put into the hospital and again given ether. Reopened along the incision of the New York operation. Dissected up the periosteum. Found the surface bone soft, the sinus obliterated on the periphery over toward the eye, but at the centre soft and containing punky, purulent material. No fluid pus. Cut away the bone almost the entire length of the sinus outwardly as far as over the supraorbital foramen, but not injuring it. Just about the centre of the forehead found one cell containing a little pus. At first I thought this cell led into the right sinus, but it only led to the median partition, which was a little to the right of the exact centre. Then dissected downward, opening one large cell leading to the nose, which contained more or less soft material. Almost the entire front wall of the sinus was taken off, leaving a little bit overhanging so as to retain the ridge. A large opening was made down into the nose and all ethmoidal cells destroyed, and a large size gauze wick was put in. Apparently sound bone left everywhere. Compared with the preceding operation of three or four years ago the sinus was smaller, showing a portion of it to have filled in with solid bone. The centre had evidently remained open and had been reinfected through a small tract, probably from the nose, which opening had not been obliterated.

The after-history of the case is uneventful. There was some pain and photophobia, but no iritis. Cavity was kept firmly packed with iodoform and balsam of Peru gauze, the external opening being maintained until absolute obliteration took place, which, so far as is known, is the case. On September 10, 1904, there was apparently complete healing. There was no discharge from the nose. The sinus was completely closed in. Twelve months have since elapsed without any further trouble. As in all of the other operations there was repeated breaking out of pus from the external wound, and as there has been none this time since discharged, I feel that the case has been completely healed.

Criticism. This case illustrates very well the necessity of complete obliteration of the sinus, as there had been on previous occasions two apparently complete cures after the two former operations, and after the last no recurrence for three years. As,

however, the centre of the sinus had not been entirely obliterated and there was still some communication with the nose, there was a chance for reinfection, which took place. The mistake which I made in the earlier instances was in allowing the external wound to close, so much so as to be unable to work through it satisfactorily without being satisfied that every bit of the centre was filled with firm bone before so doing.

Case XIV.—Mrs. B. Patient seen by Dr. S. on January 13, 1905, complaining of pain in the region of the right eye and right frontal. Next day right eye bulged very much forward. Next day bulging still the same. Pain rather diminished. Considerable pus discharge. Seen by Dr. J., an oculist, who thought the condition was probably frontal. Seen by me on January 16th. Examined carefully in bed. Slight frontal sinus tenderness; no pus in the nose; right eye protruding very markedly. Deemed it advisable to open the right sinus as an exploratory measure.

Admitted to the hospital. Temperature normal; pulse 68. Opened sinus in the internal angle under the ridge. On opening found some grumous-looking pus, not especially great in quantity, which welled forward but was apparently not under much pressure. Entire sinus did not extend very much upward, but a probe easily went down in the region of the orbit and behind it. Enlarged the bone incision with cutting forceps so as to take away a portion of the nasal process of the superior maxillary. Made large opening into the nose, which opening was apparently closed, as a probe would not go through it at first. Extended bone incision upward, and removed major portion of the anterior sinus wall, curetted out the entire mucous membrane, which was degenerated. Found that there was a good-sized opening into the opposite sinus at about the root of the nose. Opened the left sinus in a similar manner, making incision through the opening downward about as far and upward toward the notch of the supraorbital nerve, but not as far as this. Good-sized opening made into the nose, large enough to admit very large curettes. Scooped out all the degenerate mucous membrane. Packed both sinuses with gauze.

January 19th. Dressing not done for three days. Temperature has been normal. Practically no pain. On removing the dressings no sign of pus. Eye has gone backward to its natural position to quite a degree. Is not yet quite normal.

22d. Changed the dressings. Not much attempt at healing as yet.

27th. Some pus discharge on the dressings, which have a little odor at the end of two days. Right eye has resumed nearly its natural position. The only swelling seems to lie in the partition between the two sinuses curetted. There is free drainage into the nose. Patient has normal temperature; is feeling well.

28th. Is beginning to form granulations on the floor of the sinus all around. Sinuses do not extend upward a great distance. Cut

away small amount of bone with the Kerrison forceps. Patient's general condition all right.

March 31*st.* Considerable degree of filling in has occurred. Sinuses now very much smaller. Do not extend outwardly more than half an inch from the external opening. Right one extends upward a short distance. Left one practically filled except for the centre. Eye apparently normal.

June 24*th.* Left sinus entirely filled, right one open about the size of a large pea, still communicates with the nose. Now filling rapidly. Patient has been at work for several weeks. Scar on the left side inconsiderable.

July 15*th.* Complete healing; only slight scar.

While the treatment in this case lasted a considerable length of time, from the middle of January until the last of June, which length of time might be considered as a fair criticism of the method, it must be remembered on the other hand that each sinus was of large extent and that complete obliteration has been obtained without scarring to any extent.

The case which follows was of peculiar interest to me because it is the only one I have ever seen in which the empyema of the frontal sinus produced an external fistula. It also shows that a considerable degree of relief and comfort and, from the patient's standpoint, almost a cure can be brought about by merely improving the drainage.

Case XV.—W. F. T., aged sixty years. Came November 26, 1904, on account of a sore in the external nose about midway between the bridge and the angle of the eye on the right side, which was discharging pus. This had lasted for several months. He had been treated by two ophthalmologists for abscess of the tear duct. Examination with the probe showed this space to have no relation whatever to the tear duct but to lead upward into the frontal sinus and downward into the nose. Was able to syringe directly from without into each cavity. Examination of the nose showed the whole condition to have been due to a polyp or bullous-like formation of the anterior end of the right middle turbinate, obstructing the natural outlet from the sinus. The sinus was washed out several times through a cannula. The anterior end of the right middle turbinate was then removed, and as the symptoms were disappearing he did not wish any radical operation done.

December 3*d.* Rewashing of the sinus brought away dark mucopus, but the fluid pus seemed to have disappeared for the time being, and the external wound had healed, although the probe easily traversed the region around the sinus. There was more or less bare bone around the opening from the external nose. Radical operation was urged but he did not feel as though he could afford to lie up for permanent operation.

January 31, 1905. The external wound seems to have permanently healed. The discharge now comes entirely down through the nose and he is having no conscious trouble with it. The case is by no means cured, but as it is not troubling him any he declines to have anything more done.

Careful examination of the case showed that he had presumptively had a chronic sinus trouble for a good many years, the pus discharge being denominated as merely catarrh, and up to the point where the internal drainage was interfered with and the pus broke through externally, he was not conscious of much if any trouble. This case illustrates the fact that many persons will stand a pus discharge from some one of the accessory sinuses without being conscious of very much trouble for many years, and only seek relief when some concurrent inflammatory condition blocks the outlet and causes absorption of the pus. In this instance the patient thinks that my operation of the removal of the middle turbinate has cured him, whereas, as a matter of fact, I have only made the drainage from the cavity better.

Of the cases cited those operated upon, namely, Cases V. to XIV., inclusive, briefly analyzed show the following results: In 7 the sinus was opened at the inner angle of the eye, nasal drainage established, with subsequent closure of the external wound after a varying period, the so-called Ogston-Luc method or some modification of it. Of these 2 showed a cure without recurrence, 4 had one or more recurrences with final cure, 1 certainly by obliteration, the others probably; 1 is still under treatment but is practically cured, but has had more or less recurrence. Three cases were treated by the method of obliteration, and of these there have been no recurrences. The cases which showed recurrence were all of them accompanied by more or less ethmoidal suppuration or occurred in connection with maxillary antrum suppuration. In the paper by Logan Turner already referred to he states that in 62 per cent. of 172 cases, histories of which were examined by him, the ethmoidal cells were implicated, and that in 55 cases operated upon by the Ogston-Luc method there were 33 cures and 22 failures, showing probably that the successful cases were those in which there was little or no involvement of the ethmoidal cells, while the cured cases were those of pure frontal sinus trouble without ethmoidal involvement.

My own statistics are of course too few of themselves to generalize from. In the main, however, it would seem to me that the cases in which the simple opening and drainage of the sinus have been immediately successful were those in which the ethmoids were involved either but little or not at all. The cosmetic results have been good in all the cases, even those where there were several recurrences.

Conclusions. What then shall be our attitude with reference to the treatment of chronic frontal sinus suppuration? What operation would seem to offer the best chance for the patient? Certain things must enter into the consideration of each individual case, such as the length of time the suppuration has lasted, the degree of discomfort, the probable danger to life, and the age, sex, and social condition of the patient. The intranasal method of treatment should certainly be tried if it offers any chance of a permanent cure or of sufficient degree of relief to satisfy the needs of the patient. If persisted in for a reasonable length of time without result the question of external operation should be considered.

Of the external operations, viewed in the light of the past and present experiences of the majority of rhinologists, what is the one to be chosen? In simple suppuration, not long continued, I agree with the conclusion of Luc as referred to by Logan Turner, that in cases where the dimensions of the sinus are limited and the frontonasal canal is roomy and the ethmoidal labyrinth free from suppuration, and where the inflammation has not lasted any considerable length of time, the simple operation of opening and draining the sinus, with closure of the external wound, will effect a satisfactory cure. In all other cases, especially those associated with ethmoidal trouble, accompanied by the formation of polypi and the probable implication of the sphenoidal or maxillary sinuses or both, it seems to me, in the light of my own and the experience of others, that some form of the obliteration method is the operation to be chosen. Whether it shall be that which I have myself performed, namely, the taking away of a sufficient portion of the anterior wall of the sinus below the supraorbital ridge to gain access to the sinus, and so much of the floor of the nose and the nasal process of the superior maxilla as may seem necessary, followed by a prolonged packing, a method which, while reasonably certain to effect a cure, necessitates a disagreeable and long-continued amount of after-treatment if the sinus be at all large, especially long and disagreeable if both sinuses are affected, or the method of obliteration described and followed by Dr. Coakley, namely, the taking away of the anterior wall of the sinus above the supraorbital ridge with immediate obliteration and closure of the frontonasal duct, partial suture of the skin opening, with packing of the cavity until obliteration takes place, this being materially aided by the partial sinking in of the soft parts, or the more radical and much discussed operation of Killian, must be determined by the individual case, the social status of the patient, and the individual skill and proclivities of the operator. The decision in many cases can hardly be made prior to the time the patient is on the table and the sinus opened, as, although transillumination gives us considerable information in many cases, it frequently does not absolutely show the extent of the sinus, hence the operator should

be sufficiently conversant with all the methods of treatment so that he may at the time of the operation be prepared to do that one which, in the light of his knowledge, then and there offers the best results for his patient. The question of the cosmetic result must be carefully considered, as cosmetic results which would be considered good in Germany would not be so considered here.

The published statistics of Killian's operation seem to show it to be satisfactory in its results, at the same time not all of the cases showed immediate cure, and in some of them there was more or less pus discharge for a considerable length of time thereafter. Killian's operation is also not wholly devoid of danger, as Freudenthal has recorded cases where he found the dura instead of the sinus in opening up above the bridge after the method of Killian. This of course could be obviated by invariably opening against the inner angle of the eye, where, if any sinus is present at all, it will be found. Of the cases reported as cures I think some of them may justly be doubted whether they are cures or not, as so long as the discharge from the nose has not entirely stopped the case can hardly be considered as an absolute cure, even though the patient feels no discomfort. This may be illustrated in the last case reported by myself where the patient regards himself as cured, although that is far from being the case. Better drainage does not necessarily mean a cure, although it means relief.

Another operation has recently been brought to my attention by Lothrop. This is a modification of the Ogston-Luc operation, which seems to have all the advantages of the latter without the danger of recurrence of symptoms of obstruction. At the same time it possesses the advantages of the Killian, but is not followed by the deformity which this operation produces. A short incision is made under the inner end of the eyebrow so as to approach the orbital floor of the frontal sinus. The sinus is opened by this route and the whole of the nasal floor of the frontal sinus is removed chiefly by means of curettes, thereby taking away all the ethmoid cells situated in the vicinity of the ostium frontale, limited externally by the inner wall of the orbital fossa and internally by the inner wall of the lateral mass of the ethmoid, a part of which is removed. This opening embraces, therefore, as great a space as the anatomy of the parts will allow. The anterior wall of the sinus remains intact. Lothrop states that none of these operations have been followed by obstruction and that drainage is free.

It is also fair in considering any report of cases to assume that in many patients it may be impossible to get a perfect cure, as the anatomical conditions and pathological changes militate against this and do not allow the operator with safety to his patient to remove a sufficient amount of tissue. I think the operator should feel fairly well satisfied with his results when he has got his patient free from recurrence, free from annoying nasal dis-

charge, free from pain, even though there may still continue to be a moderate amount of ethmoidal mucopus which, in the majority of instances, will not be noticed by the patient as anything more than ordinary nasal discharge.

The whole subject of nasal suppuration demands more consideration from the general practitioner than it has yet had, and in view of the fact that nearly every winter epidemics of grip bring about attacks of acute sinusitis, many of which can be prevented from becoming chronic under proper methods of treatment, it becomes the duty of members of societies such as this, by their written and spoken word, to educate the average practitioner into the need of proper nasal treatment for these conditions in order that they may be recognized early and cases of acute sinus inflammation prevented from becoming chronic.

THE LARYNX IN TYPHOID FEVER.[1]

By Chevalier Jackson, M.D.,
of Pittsburg, Pa.

The vital importance of this subject is better stated by and comes with better grace from a practitioner of internal medicine. Dr. John W. Boyce wrote to the writer as follows:

"The question of great interest is whether it is possible for these affections to cause a fatal termination without such symptoms as shall plainly indicate the nature of the occurrence.

"Theoretically, it seems impossible that a man should choke to death without the conspicuous symptoms of asphyxia, particularly cyanosis; but observation has convinced me that it is only too true; that these cases may sink away as peacefully as a baby goes to sleep, and that one may not in the least suspect what is happening. I am convinced that in times past such has been the case, where I attributed the death to collapse. In the first case of this nature that I ever recognized (though it is scarcely possible it was the first case I ever saw) a fairly acute resident physician had seen the patient, and had no idea that his dyspnœa was in any way an important matter. The case was taken to the operating room for tracheotomy as a precautionary measure, not with the idea that the operation was needed but for fear it might come to be during the night in the absence of a physician. While we were making leisurely preparations for a systematic operation I accidentally noticed that the patient had stopped breathing. Fortunately the surgeon was

[1] Read at the meeting of the American Laryngological, Rhinological, and Otological Society, Boston, June, 1905.

standing there with knife in hand and was able to stab the patient on the instant. There would not have been time to send to another room for an instrument; as it was, artificial respiration was performed for fully ten minutes after the trachea was opened before the patient could be trusted to do his own breathing. In the last case I saw the patient was rapidly sinking into apnœa, which certainly would soon have been fatal had we not been watching him especially on this identical point; and we had been put on our guard by a slight change of voice, not to hoarseness, but to an aphonia, which I have been accustomed to attribute to relaxation of the vocal cords from extreme weakness and not to lesion. An interesting feature in this case was that the blood pressure as determined by the sphygmomanometer rose when the trachea was opened instead of falling, as we might expect when asphyxia is relieved. Yet there is no doubt whatever that this patient was in desperate need of air before the operation."

LITERATURE. Instead of a review of the literature, which space forbids, a bibliography is appended. The statements below differ from those in the literature radically in many points. The writer can only say that the following is a record of clinical observations as carefully made and recorded as limited time would permit.

The great prevalence of typhoid fever of the severest type in Pittsburg at all times afforded good opportunities for study. The following observations were made in the wards of the Western Pennsylvania Hospital.

In weighing these observations it must be remembered that they were all made on hospital cases which on admission are characterized by foul oral sepsis, severe toxæmia, and an aggravated type of disease due to neglect, overexertion during the early stages, unregulated diet, unhygienic surroundings, etc.

Three hundred and sixty cases were watched throughout their illness. Three separate types were observed. Subacute laryngitis, without solution of continuity of the mucosa; ulcerative laryngitis; perichondritis with or without chondrial necrosis.

LARYNGITIS. Subacute catarrhal inflammation of the laryngeal mucosa was present in 227 (63 per cent.) of the 360 cases. It seemed similar to that occurring as an independent affection, save that the surface was at all times freely bathed with secretion, much of which came from the deeper air passages.

That the larynx should participate in the general catarrhal state of the various mucous surfaces, especially the respiratory surfaces, seems natural. Were it not that a few cases develop more serious and possibly fatal lesions the subject might not merit the two years' study here recorded.

ULCERATIVE LARYNGITIS. *Frequency.* The frequency in private practice is unobtainable, but it must be much less frequent than in hospital experience. No reliable records of observations, system-

atically made on a large scale, are to be found in the literature. The frequency might be supposed to vary in different epidemics, different countries, and in different conditions and environment.

Number examined	360	cases.	
Ulcerative laryngitis in	68	"	(18.9 %)
Started as abscess of larynx in	4	"	(5.9 ")
Required tracheotomy	8	"	(11.8 ")
Perichondritis with necrosis in	6	"	(8.8 ")
" without necrosis in	11	"	(16.2 ")
Abscess from perforation in	2	"	(3.0 ")
Ulcerative trachitis in	9	"	(13.2 ")
" " with perforation and emphysema in	1	"	(1.5 ")
" " with abscess thyroid gland	1	"	(1.5 ")
Pus foci in remote locations in	15	"	(22.0 ")
Associated with acute purulent otitis media	11	"	(16.2 ")
" with leukophlegmasia	6	"	(8.8 ")
Died	4	"	(5.9 ")

BACTERIOLOGY. The typhoid fever bacillus, unaided by other organisms, has been proven to be pyogenic by a number of observers, though it probably is not often active as such without the aid or subsequent activity of pyogenic cocci. It has been found by various observers in abscesses in almost all tissues of the body, and by these observers is regarded as a prime factor. To the writer it seems reasonable to suppose that, excluding one infection (syphilis), the conditions of the soil are more active factors than the seed in any infection. In typhoid fever we have the conditions of soil for the rapid development of pus cocci. In the cases examined the bacillus typhosus was found in the saliva and in the mucosa, but not in the submucosal abscesses. It is not argued from this that it could not have been the prime factor next to conditions of soil. It is more likely, however, that the ever-present pyogenic cocci found a foothold in an abrasion of the mucosa or in an inflammation of the mucosa due to typhoid bacilli lodged from the blood flowing through the mucosal vessels; or a low resistance of cells or juices due to toxæmia; or to a necrosis of the mucosa from anæmia due to embolism or thrombosis, or to an anæmia from any other cause. Or the typhoid bacillus by causing a non-inflammatory tissue necrosis may have prepared the soil for other more active pyogenic bacteria.

The bacillus typhi abdominalis was found in the throat, both on and off the lesions, in secretions and mucosa. It was not found in any greater numbers, however, in cases with throat lesions than in those without. Pus cocci were, however, much more numerous in the cases with throat lesions, after, not before, the ulcerations developed. Of the 4 cases of abscess 2 were found pure streptococcic, 1 pure staphylococcic, and 1 these two mixed.

In one case tracheotomized for stenosis, a subglottic abscess communicating with the necrotic first tracheal ring was found. Pus withdrawn from this abscess with a hypodermic syringe before evacuation proved the pus to contain a pure culture of streptococci. Careful search showed no Eberth's bacilli.

Of the 11 cases of ulceration that also had acute suppurative otitis media it is a striking fact that in every case the same pyogenic organisms were found to preponderate in the laryngeal lesions. Thus of the 2 abscess cases that also had ear suppurations streptococci in pure culture were found in both. In the other 9 the laryngeal ulcerations, being superficial, were necessarily mixed infections, but the preponderating organisms corresponded to those of the ear. Sloughs examined microscopically in 6 cases swarmed with staphylococci, in 4 other cases with streptococci, neither being in pure culture, but mixed with other cocci. Klebs-Loeffler bacilli were found in 1 case only in this gray slough, and were not found in one case where there was an exudation in the trachea.

The following pathogenic organisms were found in the sloughs from the laryngeal ulcers: streptococcus, staphylococcus aureus, staphylococcus albus, pneumococcus, and bacillus diphtheriæ.

Laryngoscopic Appearances and Clinical Pathology. Anyone who has watched these cases can make a diagnosis of typhoid fever by the laryngeal appearance and the odor of the breath, which is peculiar, rather offensive, but not putrid. It is as distinctive as that of diphtheria.

Laryngoscopy is not difficult to one accustomed to work, as every laryngologist should be, with the patient in the recumbent position. A self-illuminating laryngoscope is convenient. The secretions should be expectorated and the throat cleared by the patient if possible. The tongue must be handled gently, and care taken that its epithelium does not come away with the towel. There are very few cases in which a good view of the larynx cannot be obtained. Œdema and nothing else may be the picture, the ulcers being hidden.

In one case three blebs were noted, one on the posterior surface of the epiglottis, another on the left arytenoid, and one in the posterior commissure. They contained sanguinolent serum and were followed by ulcers which differed in no way from the ulcers originating as sloughs.

In one case a pseudomembranous exudate lined the larynx and trachea as far as could be seen. This could be wiped away and did not leave a bleeding surface.

Four cases of purpuric spots on the laryngeal mucosa were observed. None of these were followed by ulceration. One of them had submucosal hemorrhage, and all had purpuric spots in the mucosa and skin.

Trachitis without ulceration was noted in 126 cases, though many of these were exceedingly mild. In 2 cases the trachea during life was seen to be ulcerated. One of these ulcers perforated, producing emphysema. In 2 cases tracheal ulcers were seen with the bronchoscope after death.

Of 68 cases of laryngeal ulceration the sites were as given below, a few cases having as many as four ulcers, and some cases having large ulcerations covering more than one location.

Epiglottis	42	Anterior commissure	4
Aryepiglottidean folds	22	Infraglottic region	3
Interarytenoid space	18	Trachea	4
Arytenoids	10	Bifurcation	2
Ventricular bands	7	Bronchi	2
Ventricle	5	Vocal bands	1

It is remarkable that the frequency of involvement should be so nearly in direct relation to the distance from the mouth with the notable exception of the vocal bands, which seemed quite immune. The one case in which they were ulcerated had a specific history and cleared up promptly under specific treatment.

As previously stated the larynx of practically every typhoid fever case was the seat of a catarrhal inflammation, and this did not differ from the acute laryngitis with which we are all familiar. In 5 cases this process seemed to become greatly intensified and the mucosa became diffusely crimson in color, so great was the engorgement. Minute vessels ruptured, apparently from coughing. These hemorrhagic spots later became the site of ulcers in a few cases. In many cases ulceration apparently was not preceded by any diffused increase in the intensity of the laryngitis.

Of the 68 ulcerative cases, 18 had ulcerations of the linear type, 6 were Y-shaped or branched, and 5 were wedge-shaped. Some of these cases also had ulcers of the usual rounded or oval outline.

In ten instances the development of the ulcers was watched from their incipiency. A swelling appeared over an area of mucosa, usually elongated, sometimes linear in shape, and from 2 mm. to 15 mm. in length. At first the swollen area was pale, followed in a few hours by redness. At the next stage there appeared a thin grayish slough, as if the mucosa had been lightly brushed with a solution of argentic nitrate. Through this the deeper structure could be seen to be of a dark purplish color. This faded within a day to an even opaque, dirty gray slough, which separated in a period varying from one to three days, leaving a shallow ulcer, with at first illy defined edges, and a dirty gray bed dotted in a few instances and for a short time with dark-red spots. Later the edges became thickened and the bed covered with a dirty gray exudate. In some instances the ulcer became surrounded by an areola of engorged mucosa, but more often the tissues seemed normal close up to the edge of the ulcer. The depth of the ulcer seemed to be indicated by the thickening of the edges. Soon after the ulceration reached the perichondrium œdema set in. The last two statements are based upon examination from below after tracheotomy. In 5 of the 8 tracheotomized cases the ulcers had thickened edges, and the ulceration had reached the perichondrium. The other 3 tracheotomized cases were abscesses, not ulcers.

In 1 case an abscess of the thyroid gland was opened at the tracheotomy, and the first and second rings of the trachea were found necrosed. The abscess was walled off from the neck tissues but was burrowing under the tracheal mucosa, which was pushed inward, occluding the tracheal lumen almost completely. The patient recovered promptly.

ETIOLOGY. In considering the etiology, first place must be given to the typhoid toxæmia. The blood, tissue, and juice changes are of the most profound character, and this must be borne in mind in considering every other element, as it renders operative otherwise insignificant factors. Slight local trauma, or pathologic change, that in health would be promptly cured by nature, becomes formidable in the absence of resisting power.

It is an accepted fact that a particular type of complications may characterize one epidemic of typhoid fever and a different type another. In Pittsburg we have typhoid fever with us all the time and not only as an epidemic. Yet the tendency one season to preponderance of one type of complications is very noticeable. This has to be considered in weighing etiologic factors. There must be a cause for the preponderance. When the pervading type of complication is purulent and occurs in one ward or institution the natural conclusion is that it is infective and that the source is a previous case. This was particularly noticeable in the purulent complications in the Western Pennsylvania Hospital, which nearly ceased after the elimination of flaws in the technique of disinfection of wards, beds, mattresses, pillows, etc., discovered and righted by Dr. Thomas Arbuthnot. There are several weak points in any argument based on this observation. The same tendency to type is noticed in widely scattered cases in different environments. It is well to note the tendency to laryngeal implication in certain diphtheria epidemics.

One case in 68 (1.47 per cent.) occurred before the twenty-first day. Thus, 98.53 per cent. occurred after the twenty-first day. In 8 cases that died before the twenty-first day no laryngeal lesions were found. In 8 cases that died after the twenty-first day ulcerations existed in the larynges of 2. These 16 examinations were made on the cadaver with a self-illuminating laryngoscope on cases in which autopsy was not permitted, and are not included in the 360 cases systematically examined.

These facts point to the existence of an etiologic factor after the third week that does not exist prior to that time.

What are the characteristics of the third week and the following weeks of typhoid fever as compared with the earlier period? Sensitiveness to cold, a rapid, often sudden emaciation, lowest resistance of the tissues and juices, thrombotic phenomena, glandular disturbance, pneumonia, and bronchopneumonia beginning inflammatory and purulent troubles, separation of the sloughs from

the enteric lesions. The worst of the high temperature is over, but its damages remain.

The etiologic possibilities may be classed as follows:

PREDISPOSING FACTORS. 1. Dorsal decubitus.

2. Lessened cough reflex.

3. Lessened frequency of deglutition.

4. Lowered resistance and weakened phagocytosis or its equivalent, due to toxæmia, etc.

5. Increased virulence of organisms proliferating in the air passages.

6. Intensity of the general infection: (*a*) pyrexia and (*b*) toxæmia.

7. Poor blood supply to larynx, predisposing to necrosis.

8. Pretyphoid disease: (*a*) local and (*b*) general (syphilis).

9. Draughts of air, especially when patient is perspiring.

10. Hygienically bad surroundings. Crowded wards.

11. Hydrotherapy: (*a*) sponging, (*b*) tubbing, (*c*) wet-pack, (*d*) ice-pack, and (*e*) ice-bag on head.

12. Drugs.

13. Age.

14. Sex.

15. Nationality.

16. Occupation.

EXCITING FACTORS. 17. Infection *via* (*a*) blood. Typhoid fever bacilli or pus cocci in infected emboli or floating free; (*b*) abrasions from coughing trauma or screatus.

18. Thrombosis: (*a*) aseptic clot (stasis), (*b*) infective emboli (classed above), (*c*) endophlebitis.

19. Oral sepsis.

1. That dorsal decubitus can favor thrombosis by hypostasis the writer does not believe. Anyone who will look along a ward will see that the larynx of nearly every patient is higher than any other portion of his body except his face, varying slightly accordingly as pillows may be arranged. But it seems probable that the sagging laryngeal cartilages may, so to speak, pinch the mucosa between themselves and the vertebra in the supine patient.

2. Lessened cough reflex by favoring incubation of organisms and the development of more virulent cultures in the accumulated secretions is a factor.

3. Lessened deglutition may be a factor in a similar way.

4. Lowered resistance.

The lessened resistance of the tissues in typhoid fever was demonstrated by Brieger and Ehrlich. Malignant œdema followed the injection of a solution of musk in typhoid fever patients, but was harmless in others.

Liebermeister years ago wrote: "If one studies closely the manifold complications and sequelæ of typhoid fever, in the living and at autopsies, he will receive the impression that in severe cases

the resistance is reduced to the minimum in all organs without exception, and that there is an extraordinary tendency to the destruction of the tissue." This expresses the clinical aspect of the predisposing factors. It is a condition probably brought about by the action of the toxins on the tissue cells, and is evidenced by the rapid formation (often in twenty-four hours) of large abscesses in various parts of the body. That the vulnerability of the respiratory mucosa should be similarly increased is a reasonable conclusion, while it is, more than other tissue, exposed to pyogenic cocci.

During the time these observations were in progress careful watch failed to find laryngeal ulceration in any other than typhoid fever cases with one notable exception, a case of pulmonary tuberculosis, which was sent in through error and could not be got out. Laryngeal ulceration developed in this larynx while in a room just off the typhoid fever ward. The ulcers were not tuberculous and could not be distinguished from those occurring in typhoid fever, except that the typhoid fever breath was lacking.

5. The possibility of increased virulence was not investigated, but it is deserving of research.

6. As stated, the chief etiologic factor is the typhoid fever. This renders the mucosa vulnerable and thus permits activity of other factors which otherwise would fail. Unlike the complications of the exanthemata, ulcerative laryngitis complicating typhoid fever bears as to the likelihood of its occurrence, its course, and its termination, a close relation to the severity of the primary disease. Under the head of frequency it was mentioned that inquiry among physicians in general practice developed the fact that those practitioners in the poorer districts occasionally saw cases of laryngeal complications, while many of those practising among the wealthy class never saw even one case. The reason for severe cases in the poorer districts seems to be the continuance at labor in the earlier stages (some men working with a temperature of 103°), and at the same time eating a general diet, often forcing food down in spite of anorexia, in the ignorant hope of keeping up the strength. Many laborers make an untreated walking case out of the early stage conditions that would put the wealthier man in bed with bi-daily visits of the physician and the constant attention of two trained nurses to enforce dietary and other restrictions and a proper regime. Another reason may be in the filthy habits and unhygienic surroundings of the poor. The importance of the bathtub in prophylaxis is unquestioned.

The writer's records show that out of 68 cases of ulcerative laryngitis 61 (89.7 per cent.) occurred in cases in which the temperature exceeded 104.2° every day for a week or more, and in 23 (33.8 per cent.) the temperature reached 105°. All other symptoms in a fair proportion of the cases kept pace with the temperature, as ndications of the severity of the case. No case of laryngitis occurred

in any case in which the temperature did not exceed 103°. In considering the temperature it must be remembered that except in a few cases not thought to require it the patient was sponged every time the temperature rose above 103°. Wet-packs, and in some cases ice-packs, were used if sponging failed to bring the temperature down.

Von Ziemssen thought the pyrexia was the most important factor in lowering resistance of tissues and advocated antipyretic treatment as the best mode of preventing laryngeal complications. As to the causative influence of pyrexia, this was disproven by the writer's records. Of 10 cases allowed (for other reasons) to go without sponging there were 2 (20 per cent.) developed laryngeal ulcers. This is about the same as the record of all cases, 68 out of 360 (18.8 per cent.). So that it would seem that it is the toxæmia that lowers the resisting power, and that the pyrexia is but an index of the toxæmia.

As to the effect of antipyretics other than hydrotherapy no records were obtainable, as none other is used by the staff at this hospital.

7. The etiologic possibilities of poor blood supply must be considered in the light of pathologic stasis.

8. Local diseases of the upper air passages were faithfully recorded but seemed to have no bearing except on three points. Of 68 ulcerative laryngeal cases, 10 (14.7 per cent.) were mouth breathers, 13 (19.1 per cent.) had chronic laryngitis, and 66 (97 per cent.) had a foul oral sepsis, with foully carious teeth; and of the 6 cases of perichondritis, 3 (50 per cent.) were hucksters, with a chronic laryngitis when admitted.

Several observations seem to point to syphilis being a factor in the etiology of ulceration. Histories, proverbially unreliable, were of even less than usual aid, because of toxæmic stupor, lack of an interpreter, which often rendered it impossible to separate syphilis and gonorrhœa.

Giving the record, however, for what it may be worth, specific history was: positive in 8 cases (11.7 per cent.), negative in 41 cases (60.3 per cent.), unobtainable in 19 cases (27.9 per cent.).

Specific treatment was of prompt and decided benefit in nearly all cases. But mercuric preparations and the iodides are good excretory stimulants and would render good aid in whipping up the overworked excretory apparatus and thus help to rid the system of typhoid fever toxins. This weakens the test value of therapeutics. In general syphilis favors suppurative conditions; so does typhoid fever. The concurrence of the two probably intensifies the tendency.

9, 10, 11, and 12. A chart of the beds showed that one bed located close to a door was occupied by seven successive cases of ulcerative laryngitis. No other one bed had more than two to its credit. This

door opened on to a short, draughty corridor which led to a room filled with scrubbing sinks, sewer connections, and water-closets. This corridor, on account of defective plumbing (since refitted), was always overventilated, which caused a draught to strike the bed in question. The proportionate share of the defective plumbing, the draughts of air on the sweating patient, and the defective sterilization of bedding which existed there the writer is unable to state, for all have since been corrected; and while acknowledging a weakness for experimental research, he could not, of course, endorse the reinstatement of these horrors one at a time to determine the share of each. He prefers to put them all down as etiologic factors of the first importance.

Overcrowding of wards is certainly an etiologic factor.

In regard to the effect of a draught of air he must acknowledge his inability to demonstrate wherein a draught of air on a perspiring skin differs from a cold wet-pack. If exposure to draughts of air under certain conditions may cause laryngitis in the well, why may they not so act in the typhoid fever patient?

It is Dr. John W. Boyce's practice to omit sponging in certain cases, no matter what the temperature.

He also does not advise the ice-pack. He stimulates and feeds sparingly. Other members of the staff feed and stimulate more freely, the stimulants being chiefly strychnine and whiskey, and some of them use ice-packs freely. Various drugs such as acetozone and guaiacol were tried extensively by Dr. Lawrence Litchfield. Some patients liked an ice-bag on their heads and were allowed to have it constantly, while others objected to it and it was not used.

Detailed tables would be too voluminous, but the result of observations of all these differences in treatment and management led to the conclusion that hydrotherapy, including ice-packs and head ice-bags (tubbing not being used); drugs, including stimulants, and foods and feeding are absolutely negative as etiologic factors in ulcerative laryngitis, with the single exception of the ice-pack, which had some evidence against it.

13. The age of the patients with ulcerated larynges ranged from sixteen to fifty-five years, but 71 per cent. of them were between the ages of twenty and thirty years.

14. In the female wards 3 cases of ulcerative laryngitis developed in 60 cases of typhoid fever. In the average the typhoid fever cases in the female wards were of a less degree of severity than those in the male wards, probably on account of the earlier taking the bed in the initial stages and other recognized causes. These 3 were very severe cases. As this simply brings us to the severity of the general infection, it cannot be said that sex is a factor.

15. The bearing of nativity on the etiology is interesting. Of 68 cases of ulcerative laryngitis the following were the nativities:

Arabia	1	Russia (Northern)	2
Austro-Hungary	25	Slavonia	...
Ireland	2	Syria	1
Italy	21	United States (negroes)	3
Poland	4	" " (whites)	2
Russia (Southern)	7		

The preponderance of the natives of Southern Europe is interesting, though environment and the fact that they now constitute our laboring class must be considered.

16. *Occupation.* It is worth noting that 3 cases of perichondritis occurred in "hucksters" who call their wares loudly from a wagon in the streets. They had severe chronic laryngitis with hyperplasia and dilated mucosal capillaries on admission.

17. *Infection.* There is much argument in the literature to show that the laryngeal lesions are true typhoid fever glandular involvements similar to the intestinal lesions. Local infection of the lymphoid tissue of the larynx by the bacillus typhi abdominalis is of course a possibility, but if so, in view of the cases here recorded, it must occur late when immunity is being developed.

Of the 68 cases but 1 (1.47 per cent.) occurred in the first week. It was a diffuse inflammation starting in the lymphoid tissue of the larynx. This case was probably a true typhoid fever inflammation of the lymphoid tissue of the larynx, though the local bacteriologic examination was negative. This was, however, the only case that could be so classed. The other 67 cases all occurred so late in the disease that they could not be considered true typhoid fever lesions in the sense that the lesions of Peyer's patches are. Enteric reinfections occur at this late period and the laryngeal lesions might be regarded in this light, though such is not the writer's opinion. The outline too of these ulcers, as well as the locations, negative the assumption in the literature that the lesions are specific typhoid fever ulcerations, starting in glandular tissue. Of 68 cases, 42 (61.8 per cent.) had ulcerations on or near the free border of the epiglottis where adenoid tissue does not exist.

Local pyogenic invasion assisted by trauma seemed to account for some of the cases observed. Two chief causes of trauma are the "pinching" mentioned under dorsal decubitus and cough. Coughing induced by bronchial conditions was present in nearly all severe typhoid fever cases. This cough is, of course, an irritant to the larynx favoring abrasion as well as inflammation, though of course it is highly necessary to rid the air passages of infective secretions. A longer sojourn than in the normally active state of the cough reflex probably favors the development of a virulent culture.

Two very clear cases of local pyogenic invasion occurred. In both a stream of pus from an acute suppurative otitis media was

draining into the laryngopharynx from the Eustachian tube mouth. The ulcers were extrinsic (confined to the epiglottic and posterior cricoid mucosæ), and disappeared promptly after circumcision of the membrana tympani with adequate "wick" drainage through the external auditory meatus.

18. The period and the suddenness of the onset, together with the laryngoscopic appearances as already described, especially the linear wedge-shaped and branched outlines, point to a local thrombosis as one of the chief exciting factors.

As to the etiology of this thrombosis, whether it be primarily an aseptic clot from stasis, or from an endophlebitis, or an endarteritis, or an embolus (aseptic or infective), the writer has only two postmortem findings to offer. Two cases with the linear and branched ulcerations had multiple infarcts in lungs and kidneys. These were the only autopsies. Of course the arteries of the larynx are not "terminal arteries," and thus are not subject to infarct exactly as those of the lungs and kidneys are, but arterial occlusion would by ischemia favor limited necrosis and bacterial invasion.

An embolic etiology would require an original focus in the lungs or left heart or in the periphery proximal to the laryngeal arteries. It is to be regretted that the occurrence of endocardial and other possible lesions bearing on embolic etiology were not recorded.

Several arguments against the thrombotic pathology might be advanced. The number of cases of ulcerative laryngitis associated with leukophlegmasia was small (6). Yet each of these had ulcerations of the linear type. It might be argued that femoral phlebitis and thrombosis is a common complication in private practice, while laryngeal ulceration is rare. And certainly the writer must acknowledge that he has seen but one case of laryngeal ulceration outside of a hospital. But it must be remembered that he has never had an opportunity of systematically observing the throats of typhoid fever cases in private practice. It may be frequent yet, as pointed out by Dr. Boyce, unrecognized, even though it actually be the cause of a fatal termination.

19. Oral sepsis is closely related to infection. Its part seems so self-evident as to require no proof.

Glossitis was present in 2 cases and in 1 case through extension by continuity was the cause of perichondritis, chondrial necrosis, and abscess with subsequent ulceration.

Symptomatology. The writer considers it his duty to state as a preliminary that all the classic symptoms of laryngeal trouble may be absent, or so slight as to escape notice, being masked by the general malady. Categorically the local symptoms that may be noted are: pain and tenderness, stridor, dyspnœa, dysphagia, odynphagia, dysphonia, odynphonia, hoarseness, croupy cough, and external swelling over the thyroid cartilage. Expectoration is masked by bronchial expectoration.

The general symptoms occasionally seen are recrudescence of pyrexia and extreme prostration with symptoms of approaching death by toxæmia and exhaustion. The pyrexia, as in all post-typhoid fever conditions, usually partakes of the type peculiar to the general malady. In a few of the writer's observations the laryngeal complication only reached the stenotic stage so long after the general toxæmia that the dyspnœa was of the usual active type. But in the feeble state of the profoundly toxæmic typhoid fever patient it was not the symptom we see elsewhere. In 4 of the patients saved from asphyxia by tracheotomy there were none of the symptoms we are accustomed to look for—no sitting erect, gasping, stridor, struggling, violent action of the accessory respiratory muscles, cyanosis, etc. In 3 others slight cyanosis was present and some effort was being put forth, while only in a few cases was the patient making a typical fight for air.

Dyspnœa when present may be due to œdema, occlusion of the respiratory lumen by a projecting abscess, occlusion of the glottis by a fragment of necrotic or sloughing tissue, collapse of the cartilaginous box from necrosis, fixation of, or abductor paralysis of, the cords.

Hoarseness is the most frequent symptom; aphonia next.

The usual cough of typhoid fever was present in 50 (5.9 per cent.) of the 68 cases, but in only 36 of the 50 could it be said to be changed in character after the onset of laryngeal lesions. In a few of these it became quite hoarse and croupy.

The onset of symptoms, averaging all cases that had symptoms, was the thirty-first day. The onset of ulceration in all cases averaged the twenty-second day.

Diagnosis. Diagnosis is easy if the larynx be examined regularly. If reliance be placed on a watch of the symptoms not more than 1 case in 10 will be diagnosticated, and, as pointed out by Dr. Boyce, a certain number will die without the diagnosis ever being made, even in some of the instances in which the laryngeal condition is the real cause of death.

Prognosis. As to life, the prognosis is good if considered apart from the primary general infection. If watched by one skilled in the use of the laryngoscope there is little danger if the suggestions given under treatment be followed. Practically, however, it is not possible to separate the prognosis, and when it is considered that the laryngeal lesion appears in practically always the profoundly toxæmic cases the prognosis as to life must be said to rest altogether on the patient's general condition. As a matter of statistics no case of the 68 died of either the laryngeal lesions or the tracheotomy done for their relief. Only one tracheotomized case died and that was of toxæmia. Of the 68 cases, 4 cases (5.8 per cent.) died; 1 of relapse with intestinal perforation, 2 of toxæmia, 1 of pneumonia. The mortality (5.8 per cent.) seems low, considering that all were severe

cases, but it must be remembered that the laryngeal ulceration only appeared after the patient was convalescent and had run the gauntlet of his greatest dangers. How many early fatal typhoid-fever cases would have had laryngeal lesions had they lived of course is beyond human knowledge. In 3 cases ulceration perforated into the soft tissues of the neck, but in no case was there a cellulitis or violent inflammation of the extralaryngeal tissues.

No case need die from the throat complication. The greatest danger to life is from failing to recognize the existence of a laryngeal lesion. Tracheotomy is wrongly regarded as an operation dangerous to life. It has been credited with too many deaths that were justly attributable to the condition calling for it. Of course, as it is the profoundly poisoned cases that get ulcerative laryngeal lesions the prognosis as to life must be guarded, but not because of laryngeal dangers.

As to Voice. In 45 out of the 68 cases (66.2 per cent.) the ulceration healed without alteration in voice or structure, no visible scar remaining. Permanent deformity, without loss of voice and without stenosis, resulted in 8 cases. Stenosis requiring surgical relief to enable the abandonment of the tracheal cannula resulted in 5 cases, all of which recovered good, useful voices of rough quality and uncertain pitch. Cicatricial stenosis can always be avoided if the suggestions under treatment be followed. If it should occur from neglect of early tracheotomy it can be cured with good voice by thyrotomy and prolonged intubation. As to paralysis and fixation, they cease to affect the voice ultimately though their stenotic effects cease only by operative measures.

TREATMENT. In those cases of ulcerative laryngitis that are primary specific typhoid fever lesions, and also those that are of hæmatogenous origin, whether by embolism, thrombosis, or blood-borne pyogenic or specific typhoid fever organisms, prophylaxis is hopeless. But even in these cases the writer does not hesitate to say that if closely watched and judiciously treated no ulcerative case need go on to chondrial necrosis and laryngeal stenotic deformity. These occur in the lately discovered cases and in those cases in which a foul state of oral sepsis, an untreated mucosa, and a long postponed tracheotomy favor a great destruction of tissue.

Prophylaxis then must seek to hold in check the mixed infections that run riot.

The Room. The air should be kept as free from pus organisms as possible, and all cold draughts of air should be avoided, especially if the patient is perspiring. Mattresses and pillows, the entire room in fact, should be sterilized by thorough fumigation. In hospital wards overcrowding must be guarded against.

The Patient. Careful and persistent attention to oral antisepsis (asepsis being impossible) is the first consideration. A dentist's aid should be sought, a daily brushing of the teeth with a soft brush

if the gums be tender, followed by swabbing with a one-half of 1 per cent. carbolic acid solution, with borax, sodium bicarbonate, menthol, alcohol, and camphor-water is the most agreeable and refreshing as well as cleansing agent. The patient should be encouraged to clear his throat frequently. All food and water should be sterile. From a laryngologist's viewpoint the ice-pack should not be used unless deemed necessary to prevent a fatal degree of hyperpyrexia.

The writer does not commend the spraying of the nose except where there is a tendency to accumulation of dried or crusted secretions. Usually the natural posterior drainage of the nose, favored by dorsal decubitus, is sufficient to keep the nose clean.

In all the prophylactic measures one thing must be clearly borne in mind, namely, anything that excites or annoys the patient is a detriment to his general condition, so that we must be very sure that what we do is of unquestionable value. The regular toilet of the mouth is agreeable to the patient and is certainly a good thing from the laryngologist's as well as the attending physician's viewpoint, and need not annoy the patient.

In the luetic cases specific treatment yielded prompt results. In other cases considered able to stand it the same treatment yielded good results, probably by acting as an excretory stimulant assisting in the elimination of toxins.

Inhalation of benzoin from hot water is useful. The room may be steamed with water kept boiling to which tincture benzoin compound is added.

In acute œdematous stenosis adrenalin solution 1: 1000 (or any of the similar preparations) as a spray is useful to gain time and may tide over a danger point, but it may become a danger if it lead to too long a postponement of a tracheotomy.

Intubation is useless in œdema, worse than useless in abductor paralysis. If used for the latter, glottic closure will terminate the case promptly should the tube be coughed up.

Of the 5 cases of cicatricial stenosis 3 have been cured by thyrotomy, followed by intubation. They have good though hoarse voices, the tracheotomy wounds are healed and ample laryngeal lumen has been obtained. The method of treatment was given in a paper by Dr. Ewing W. Day and myself, read before the Middle Section of this Society in 1904. One, the last, is still under treatment, and 1 was cured by prolonged intubation with large special tubes.

If closely watched and tracheotomized early, chondrial necrosis with its attendant laryngeal deformity, stenosis, alteration of voice, can all be prevented. All lesions disappear as if by magic after an early tracheotomy. Inability to abandon the tracheal cannula indicates too long a postponement of tracheotomy.

Direct medication to the interior of the larynx is easily accom-

plished through the tracheotomy wound, though these cases seem to recover promptly without medication after the trachea is opened.

Abscesses may be incised endolaryngeally if desired, but preparations for tracheotomy should be first made, as it may become urgently needed. Personally, the writer would advise tracheotomy with subsequent incision of the abscess through the wound.

With regard to tracheotomy it is practically harmless if Schleich infiltration anæsthesia be used, if it be bloodless, if the patient be in the Trendelenburg position for the operation, and the day after. If tracheotomy be attempted with a general anæsthetic it will usually end up with respiratory cessation and a stabbing operation. Then we have added to the shock of a general anæsthetic the loss of blood, lack of time for hæmostasis. A few days ago Dr. John W. Boyce locally anæsthetized with salt solution a boy of fourteen years who had been terrified by being told (prior to admission) that he would die on the operating table. Yet he made no resistance and acknowledged no pain when the writer did the tracheotomy.

Toxæmia and also carbonic acid narcosis lessen the need for a general anæsthetic in typhoid fever cases, though no case should be allowed to go on to carbonic acid narcosis. After the respiratory centres are profoundly poisoned recovery may be impossible.

The writer here dwells at some length, as he feels strongly on this subject. The astonishing records appear in the literature of three different laryngeal typhoid fever cases which were considered too feeble to withstand a tracheotomy, yet they lived more than two days after it was decided that they had not sufficient vitality to withstand the operation. From their histories they apparently died for want of a tracheotomy.

PERICHONDRITIS AND CHONDRITIS. Perichondritis and chondritis are so intimately associated that clinically they cannot be separated.

The poor blood supply of laryngeal cartilages, lessened by the feeble circulation induced by toxæmia, and still further diminished by stasis, is undoubtedly one of the most important factors in causing necrosis.

Of the 17 cases of perichondritis, 4 started as abscesses and 13 occurred from deepening of superficial ulcerations.

The cartilages involved in the order of frequency were the arytenoids, the cricoid, the thyroid, and the annular tracheal cartilages. As to the involvement of the cartilages of Santorini and of Wrisberg it was impossible to determine clinically.

The epiglottis, while the site most frequently involved in ulceration, was in no case the seat of chondrial necrosis, probably because of its better blood supply.

The writer wishes he could demonstrate pathologically that there is a purulent diathesis. Every clinician sees and knows a type of patient in which every inflammation runs to pus. Many of these cases of abscess and necrosis seemed to be of this type.

Next to this as a cause of chondrial necrosis the writer believes that lues is a fundamental element, not so much as an active lesion as a predisposing factor.

RESUME. From laryngoscopic observations of 360 typhoid fever cases the writer concludes that:

1. Serious and fatal lesions of the larynx are much more frequent than is realized.

2. Death may occur from laryngeal stenosis without even the existence of a laryngeal lesion being suspected in the absence of laryngoscopy. If pain and hoarseness be depended upon a diagnosis will seldom be made. Pain is often masked by toxæmia. Cyanosis and dyspnœa are rare, apnœa is common.

3. Unlike the complications of the exanthemata, ulcerative laryngitis complicating typhoid fever bears as to the likelihood of its occurrence, its course, and its termination, a close relation to the severity of the primary disease.

4. The severity of the laryngeal lesion is in direct proportion to the toxæmia, pyrexia not being in itself a factor, but only an index of the toxæmia.

5. Thrombosis of laryngeal vessels in the mucosa or deeper is probably the most frequent local initial lesion.

6. Mixed pyogenic infections are the rule. Laryngeal lesions due to the bacillus typhi abdominalis are exceedingly rare.

7. Prognosis as to life is good if considered apart from the general malady. Not only the life but the laryngeal vocal and respiratory functions will be saved if a tracheotomy be done early. Death from laryngeal lesion means a death for want of an early tracheotomy.

8. Prophylaxis consists in good ventilation, without draughts, sterile bedding, oral antisepsis, sterile food, and water.

9. *Treatment.* Potassium iodide, hydrargyrum biniodid, benzoine inhalations, and oral antisepsis are the best remedies. Early tracheotomy under local (Schleich solution) anæsthesia will cure almost every case.

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In conclusion, the writer urges the laryngoscopic watch of the typhoid fever convalescent or rather defervescent.

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ANGIONEUROTIC ŒDEMA INVOLVING THE UPPER RESPIRATORY TRACT.*

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Angioneurotic œdema, as defined by Collins,[1] is a "vasomotor neurosis or an angioneurosis, characterized by the appearance of circumscribed swellings on various portions of the surface of the body and the mucous membranes, by preference the face, throat, and extremities, without apparent cause or premonition, and non-inflammatory in character."

As it affects the skin, the disease was first described by Quincke as "An Acute Circumscribed Œdema," and from his first description the disease was early called "Quincke's disease." Shortly after Strübing[2] described it as affecting the throat, calling it an "angioneurotic œdema of the throat," regarding it as a vasomotor neurosis, developing rapidly, without fever, after exposure to cold or some traumatism.

The disease is frequently described in books on diseases of the skin under the heading of urticaria. Crocker regards it simply as one of its varieties, giant urticaria. Stelwagon treats of it under a heading of its own, recognizing its similarity in many cases to urticaria, but in other cases says it is entirely or sufficiently independent

* Candidate's Thesis, American Laryngological Association, June, 1905.

of it to warrant its description under its own title. Montgomery and Hyde say it should not be confused with erythema multiforme, erythema nosodum, or giant urticaria. Kaposi and Shoemaker refer to it as a distinct disease, but closely allied to giant urticaria, with which, however, it is not to be confounded.

Osler[3] has been greatly interested in the disease, and in 1888 reported a case of angioneurotic œdema, investigating particularly the question of heredity. Since then he has made three notable communications,[4] reporting in the three papers 29 cases, and in the last one reviewing them all under the title of "The Visceral Complications of the Erythema Group of Skin Diseases."[5] In this paper, and based upon his cases, he shows very conclusively that simple erythema, erythema exudativum, erythema nodosum, certain of the purpuras, urticaria, and angioneurotic œdema, are all very intimately related; that the lesions of any or all of these various affections may, and often do, occur in one individual during the course of his disease, or even during one attack of the disease, so that at one time one observer might make a diagnosis of urticaria; in a subsequent attack another may call it angioneurotic œdema, or during another attack peliosis rheumatica. In a word, a patient observed for a number of years is likely to present at one time or another a variety of lesions, all of them, however, vasomotor neuroses, and so he prefers to classify them under the general head of "the erythema group of skin diseases." He divides the complications into two great groups, the angioneurotic and the inflammatory. To the angioneurotic belong the swellings of the fauces, œdema of the glottis, changes in the bronchial mucosa, causing asthma and the colic due to the œdema of the gastrointestinal tract. The inflammatory group are more serious and include endocarditis, pericarditis, pleurisy, pneumonia, and nephritis.

It is unfortunate that so many writers in reporting their cases have used the term "urticaria" in describing "angioneurotic œdema," this being apparent from their description of the lesions and it has led to much confusion.

While all text-books on the skin describe this disease under one heading or another, and nearly all refer to the fact that the lesions affect the tongue, fauces, and larynx, yet text-books on the nose and throat, with few exceptions, have neglected any reference to or description of the disease in any way. This is particularly so in the case of American text-books. Of sixteen of these latter, but three (Kyle, Posey and Wright, and Bosworth) make any mention whatever of the disease, and in but one (Bosworth) is there any description at all.

Case I.—J. B., male, aged twenty-four years, single, blonde, of a marked neurotic temperament, consulted me at 7 o'clock in the morning of April 4, 1893, twelve years ago, because of difficulty in swallowing and speaking, and of a swelling in the throat, which had

come on suddenly three hours before, wakening him from sleep. He had gone to bed feeling perfectly well.

The open mouth, fixed jaw, thick speech, and evident pain and discomfort in speaking at once suggested quinsy.

On examination I found a pale yellow, almost translucent, œdematous swelling of the right side of the fauces, almost completely filling this half of the faucial opening. The parts involved were the anterior and posterior pillars, the right half of the velum palati, the uvula, and the lateral wall of the pharynx of the same side. The tonsil itself was not swollen, though pushed out as in quinsy. Except for the swelling there were none of the signs of inflammation, the color being a pale yellow, the contour alone resembling peritonsillitis. The larynx was normal, except for the slight congestion of the vocal cords; tongue not swollen, but thickly coated; some earache. He complained of a slight dyspnœa, and marked dysphagia. Temperature normal, pulse 90, constipated. Has now and has had some gastric disturbance. Urine 1022, acid, no albumin. He gives this history:

About a month ago, for the first time in his life, there appeared, without apparent cause, a localized œdematous swelling, without pain or redness, just above the olecranon of the right elbow, followed the next day by a similar swelling over the biceps of the same arm. These were soon followed by like swellings in both feet after a long walk, then in both hands; in a day or two another swelling made its appearance in the left shoulder following a bruise. The next swelling was in the fauces somewhat similar to the present one, except that it was on the opposite side and was very much smaller. This, as had those in the skin, disappeared within twenty-four hours of the onset, and he had a respite for three or four days. The next was serious enough to threaten life. At this time there came on rapidly great difficulty in breathing, rapidly increasing, so that he immediately consulted the laryngologist who had been treating him, and who had the previous day applied a zinc chloride solution to the larynx. After examination he was told there was an œdema of the larynx, pale in color, and in appearance resembling a polypus, but which his physician regarded as urticarial. The dyspnœa was so great that it was feared tracheotomy might be required at any moment. However, without any local treatment beyond the use of a cocaine spray and the internal use of salines, the dyspnœa gradually diminished and the swelling disappeared entirely within thirty-six hours.

His tongue and right cheek were next affected. A day or two later, following a bicycle ride, there came on quickly a swelling of the genitals, the scrotum and penis being enormously swollen, as well as the inner aspects of both thighs, extending from the anus behind to Poupart's ligament and over the hypogastrium in front. This swelling was not painful beyond the feeling of distention; did

not burn or itch, and was not reddened. It kept him in bed, and disappeared in forty-eight hours. From this time until I saw him he had several minor œdematous swellings on the hands and feet.

I scarified with a small bistoury the mucous membrane in a number of places, causing immediate relief to the discomfort and distention, and a marked subsidence of the œdema. Ordered discontinuance of cocaine and antipyrine spray, which he was using, and prescribed saline purgatives. Nine hours later the swelling in the throat was almost entirely gone; pulse and temperature normal. Feels well, but there has suddenly appeared since morning an œdema of the upper and lower lids of the right eye, closing it. Two days later there was no swelling whatever in the throat, and none in the eyelids, but one is just beginning over the left mastoid.

The patient, who had come on a visit to his parents from Brooklyn the night before I saw him, now returned, and I did not see him again for several years; but from a letter a few weeks later learned that he had a few slight swellings in hands and feet, but nothing serious. In writing he called my attention to the fact that all three acute attacks in the throat followed the free use of the cocaine and antipyrine spray, which had been prescribed for nasal obstruction, of which he had been having a great deal of late.

These different swellings of the throat and of the skin were all characterized by coming on suddenly; were circumscribed, œdematous, did not pit on pressure, and there was an absence of redness, heat, pain, and throbbing, not at all resembling an urticarial wheal, excepting in the suddenness of occurrence and subsidence.

Family History. On mother's side, grandmother and her sisters had, as children, urticaria severely. One uncle has an attack of it after eating canned fruit. This uncle's son as a child had the disease so badly that "he would go into spasms." Patient's mother, five years ago, had urticaria off and on for six months, being under medical care continually for it. It disappeared after a successful perineal operation. The mother's family have for years regarded urticaria as a family disease. They are also distinguished among themselves by a peculiar gastric affection, which they designate as the "S—— stomach." No other family history of gout or rheumatism.

The attack in which I first saw this patient occurred twelve years ago. He was at that time a director of a gymnasium. He is now a physician, and in reply to a letter written him recently, he writes me, under date of April 3, 1905, that up to about two and a half years ago he had considerable local trouble in the head and throat of a catarrhal nature, but without the occurrence of the previous œdema experienced when he was under my care twelve years ago. On two separate occasions five years ago he had serious attacks of intestinal hemorrhage. These were preceded for a day or more by a feeling of weakness, depression, weight in the epigastrium, followed by a

condition of collapse resembling a moderate degree of shock. This was followed in a day or two by a profuse tar-colored stool, staining the water red, and with it much prostration for a few days, followed by a gradual recovery. For seven years, from 1896 to 1903, he had considerable intestinal trouble, the principal manifestation being the enormous accumulation of gases, which would rise into the stomach and be belched up, very little passing through the bowels. For the past two years he has been gradually improving, very little of the catarrhal trouble in nose and throat, and the gastrointestinal difficulty gradually but steadily lessening.

This patient has had the disease twelve years. He had the typical angioneurotic œdema of the skin, fauces, larynx, and nose twelve years ago, almost requiring tracheotomy on one occasion, and in the nose so badly that for a time the only relief obtained from the nasal obstruction was through the use of cocaine. In addition to the lesions affecting the skin and upper respiratory tract, he had, five years ago and again two years ago, evidences of the same disease affecting locally the gastrointestinal tract, as well as hemorrhages, probably due to purpura, from the intestinal mucous membranes.

His family history shows the influence of heredity in a marked manner. Being a physician, he was greatly interested in the cause of the various attacks, but was never able to determine the immediate cause, excepting that on two occasions he thought the attacks followed traumatism. He never discovered that any particular article of food produced an outbreak.

Case II.—A. M., aged fifty-five years, a manufacturer; of a decided nervous temperament. He has been under my observation off and on for nasal and throat difficulty for about six years. He came under my care first because of nasal obstruction, which would come on suddenly and as quickly disappear. There was no periodicity noticed about it, and it occurred at all seasons of the year; would last a few hours or days, no sneezing, and none of the usual signs of a cold in the head, nor did it act like hay fever. The peculiarity was that this obstruction came on usually during the night and by morning was gone. He also had suddenly arising swellings in the throat; the uvula and soft palate would become œdematous, and the dyspnœa would waken him from sleep, so that he was often compelled to sit up to breathe. The voice would become thick and husky at times. There was no oppression or rales in the chest. He insisted the dyspnœa was not that of asthma, but due to the swelling in the throat. The condition would usually begin to improve in a few hours, and in twenty-four to thirty-six would usually have disappeared. An examination of the nose showed at times a rather pale œdematous infiltration of the turbinates, sometimes of one, sometimes of all; but on another visit there would be no sign whatever of any thickening, hypertrophy, or œdema. The only con-

stant abnormality appeared to be a thickening of the septum opposite the middle turbinate on the right side. This thickening of the septum and the anterior end of the middle turbinate I removed, and while it improved his nasal breathing, it did not do away with or lessen the frequency of these suddenly occurring obstructions. These nasal and pharyngeal swellings would be preceded, followed, or there would occur at the same time, pale yellow swellings of the skin of various parts of the body, notably the eyelids, the scrotum and penis, the feet, and hands. These attacks would last from a few days to two weeks, and he might be as long as two months, and occasionally has gone six months without an attack. They have been recurring during the past eighteen years. He is at the present time having an attack, and a description of it will answer for all.

It is two months since the previous outbreak. A few evenings ago, while attending a banquet, the left side of the tongue began to swell; on reaching home he took a large dose of alkalithia, hoping to ward off the attack. The following morning he came to my office with the right upper eyelid œdematous and swollen, and the eye closed; it was pale, yellowish, did not burn or itch. No throat or nasal lesions, and the swelling of the tongue gone. That night he awoke, finding the nose obstructed and the throat swollen, and in the morning I saw him. There was a patch of œdema larger than a bean on the left side of the hard palate, and a pale œdematous swelling involving the uvula and the left posterior pillar of the soft palate, extending down the lateral side of the pharynx to the pyriform sinus. The mucous membrane of the left arytenoid was as large as a cherry. The obstruction in the nose had disappeared and there were no œdematous spots here. There was no pain in the throat, but merely a sense of fulness, with slightly obstructed breathing. On the skin there was a swelling under an inguinal truss and the scrotum was swollen. There is, and has been, no disturbance with digestion; bowels regular. Dr. Dutcher examined the urine and reported as follows: "Color pale amber, slightly cloudy, faintly alkaline, specific gravity 1023, no albumin; no sugar; urea, 2 per cent.; slight indican; no excess of uric acid. Microscopic examination showed mucus and an occasional epithelial cell." The œdema in the throat and skin entirely disappeared during the day, and there was no reappearance for three days, when the right upper and lower eyelids became swollen and closed the eye. This is a typical attack, such as he has been having at intervals for the past eighteen years. The swelling in the feet prevents his wearing his shoes and confines him to the house at times. The dyspnœa is often alarming, but he has never had to call a physician during the night. He says the attacks are becoming less frequent than formerly, and attributes this to the fact that he thinks he knows when they are coming on by his urine becoming more acid, and he at once resorts to alkalies. He has watched his diet during all these years, and has finally given

up thinking any specific kind of food provokes the attacks, as he has from time to time excluded almost everything without that making much difference.

Family History. He has two children, both of whom have been under my care. The son had asthma until ten years of age, and I removed the adenoids because of this ten years ago and with an apparent relief of the asthma. I have not seen him since, and his father says he has had no attacks in several years. He has never had urticaria or the angioneurotic œdema as his father.

The daughter had repeated attacks of what I thought was rheumatic pharyngitis, which recurred with great frequency. I was never satisfied with my diagnosis, and as she was under my care before her father, it did not occur to me that the difficulty might be one allied or related to angioneurotic œdema. Her father says she never had hives or his disease.

The patient had one brother, who, during the latter years of his life, suffered repeatedly and severely from attacks of what were diagnosed as "gallstone colic," and had outbreaks of a skin disease, said to have been eczema. Two years ago he fell, injuring one of his knees. One month later, while in apparently good health, after being at stool, he suddenly fell and died immediately. A searching autopsy was made, but failed to find any cause of death. An embolus in the brain was suspected, but was not found, and, as the case came into court through an action to recover from the accident company, the cause of death was thoroughly canvassed. The autopsy revealed no cause whatever from the gallstone colics from which he had suffered. In the light of the angioneurotic disease in my patient, his brother, the history of gallstone colics for years, without a discoverable cause at autopsy, and the fact that, as Osler has reported, suddenly arising œdemas occur in the brain, it is fair to question whether this might not have been such a case.

The next case appears to be a genuine one of urticaria, affecting the pharynx, larynx, and probably the nose, and shows both the similarity and the difference in the lesions and subjective symptoms of angioneurotic œdema and urticaria.

Case III.—E. R., aged thirty-five years, merchant; of a highly nervous temperament, consulted me June 24, 1904, because of what he called "a sudden swelling of the tonsils and a feeling of impending suffocation at such times." He snored a great deal at times and at others not at all. The day before consulting me he had entertained a number of men at his country house, which had made him very nervous and had drunk quite a little beer, and that night thought he would die before relief could be had because of the sudden swelling of his throat and obstructed breathing. This came on in a moment and gradually subsided, passing off in half an hour. These attacks of dyspnœa and the desire to constantly swallow have been

occurring for about five years. The first attack followed an outbreak of what he called hives all over the body, including the face and lips, which became greatly swollen. Since that first attack there have been no skin lesions. The swellings in the throat had, of late, been coming on every day or two, though at times he would go a couple of weeks, or even a month. He has been treated by a number of physicians without relief. On examination I found a deflected septum, partially obstructing the left nares, tonsils somewhat enlarged, and the uvula slightly elongated. I could discover no œdematous swelling in the throat, nor any other local trouble. He was not having any symptoms at the time of the examination. He had never had asthma or colic. The urine examined the next day was amber, clear, highly acid; specific gravity, 1030; urea, 4 per cent.; no albumin; no sugar; no indican; a very large excess of acid; no casts; no pus.

I made a diagnosis of angioneurotic œdema, prescribed alkalies and large quantities of water, and explained the nature of the trouble, and asked him to come in again on recurrence of the first attack. He did not return, however, and I did not see him again for nine months; in the mean time he had been under the care of several specialists, one of whom partially excised one tonsil, without, however, affecting the sudden swelling of the throat; another proposed uvulotomy, and the third assured him his symptoms were entirely nervous and largely imaginary. When I saw him recently he was having what he called an attack. He came to my office greatly excited; said his throat felt as though it would close up, and he could not breathe. He was greatly alarmed, and I expected to find a great deal of swelling. On examination, however, the tonsils, soft palate, and uvula and larynx were entirely normal, and I was about to conclude that my diagnosis of nearly a year ago was wrong, when I discovered, with the mirror, on the posterior wall of the laryngopharynx three elevations about the size of a split pea. They were raised above the surface, red, not at all œdematous in appearance, but resembled the wheal of urticaria as seen on the skin. Irritating the membrane half a dozen other elevations soon appeared in size from a pinhead to larger than a pea. The patient said there was no pain, no burning, but a sense of the enormous fulness, and he could hardly believe my statement of the size of the swellings. They were evidently urticarial wheals. In him they would be produced by any slight pyschical irritation or annoyance, such, for instance, as being kept waiting at the telephone, or a person with whom he had an appointment being late, or the unexpected meeting of a friend. These attacks would come on immediately, so that he could not speak for a few moments, but would clutch his throat, begin to swallow rapidly, often to the great consternation of the person with whom he was attempting to converse. An attack would begin often with the sense of burning at the end of the penis, with the

desire to urinate, and then this feeling of swelling in the throat would come on.

Family History. His father died a few years ago, and as far back as the patient can remember he suffered from colic, walking the floor nights repeatedly, suffering great pain and the belching of gas because of it. He was said to have had "nervous dyspepsia." He had never had asthma. He died after five days' illness of acute uræmic poisoning without a previous history of nephritis.

A sister of the patient has had urticaria twice during the past two years. She has had a great deal of indigestion with pyrosis.

This appears to be a genuine case of urticaria affecting the pharynx and larynx and the nose. That it may be easily overlooked is seen by the fact that within a year three different specialists who saw him failed to appreciate the condition, probably because no lesions were present when he was examined. Goodale[6] states that urticaria, although observed upon the mucous membrane of the tongue, does not appear to have been found upon the mucous membrane of the nose or throat. Certainly these throat lesions of his resemble the wheal of urticaria observed on the skin. The amount of local irritation and the subjective symptoms produced were out of all proportion to their size, in this respect greatly differing from the lesions of angioneurotic œdema, as seen, for instance, in the throats of Cases I. and II., in which there were ten times the amount of swelling and mechanical obstruction without anything like the amount of subjective symptoms.

The acute circumscribed swelling of angioneurotic œdema, as it appears in the mucous membranes of the respiratory and the gastro-intestinal tract, appears to resemble that on the skin. It comes on suddenly, in a few moments or sometimes minutes, without warning, while the patient is asleep or awake, and the symptoms it produces seem to be largely due to mechanical obstruction. In the nose, the symptoms are those of obstructed breathing, not much sneezing, and no excessive secretion, differing in this respect from hay fever, but resembling it in the suddenness of occurrence and subsidence. Doubtless many cases of this disease affecting the nose are diagnosed erroneously as hay fever, which disease it closely resembles, both in its etiology and pathology. Both are vasomotor neuroses, both are markedly hereditary and occur in persons of neurotic temperament, while the œdema of angioneurotic œdema is usually transient, lasting from a few hours to a day or two; that is not always the case. Bloodgood[7] has reported what appears to be a genuine case of this disease, which he calls "angioneurotic erythema persistens," in which the parts involved in the œdema were the mucous membrane of both maxillary sinuses and the skin of both cheeks. The œdema persisted for months. He opened both antræ through the canine fossæ, finding the mucous membrane greatly œdematous. The relief following this operation was

but temporary, the erythema and œdema recurring until neurectomy of both infraorbital nerves was done. This gave permanent relief, although the same condition appeared later on the skin of the abdomen.

Osler has also reported cases in which the œdema persisted for a long time. Some of these cases of what are sometimes called perennial hay fever may be those of this disease in which the swollen and œdematous mucous membrane of the nose is not transient but persists. Haviland Hall says: "Urticaria is frequently associated with hay fever," and there is certainly a family likeness between hay fever, on the one hand, and urticaria and angioneurotic œdema of the nasal mucous membranes on the other.

The relation of angioneurotic œdema, and, more particularly urticaria, to asthma has been frequently noted. F. A. Packard[8] reported 3 cases of his own in which attacks of asthma alternated with outbreaks of urticaria on the skin, the cough and dyspnœa being relieved upon the occurrence of the hives, and upon a subsidence of the latter a return of the asthma. The asthma was caused apparently by the presence of wheals on the bronchial mucous membrane. Packard made a thorough search of the literature, and, in addition to his own cases, found and abstracted the reports of 34 other cases in which there was a close connection between urticaria and asthma. Throughout his paper he uses, unfortunately, it seems to me, the term "urticaria" to include "angioneurotic œdema."

In a case of asthma under the writer's care twelve years ago, the patient, a man, aged sixty-five years, had had the disease for about thirty years, and had been under the care of many excellent men, but never received more than temporary relief. He was referred to me because of a possible nasal cause, and I found the mucous membrane of all the turbinates hypertrophied, gray in color, at times greatly œdematous, obstructing the nose completely, and at such times the asthmatic dyspnœa was always greatest. There were no polypi and the septum was straight. He was scarcely ever entirely free from dyspnœa during the year I saw him, and at times, both day and night, in his office or on the street, the dyspnœa was so great that he was a most pitiful object. I cauterized his turbinates as well as removed the inferior borders of both lower turbinates, giving him temporary relief only. The asthma soon recurred as badly as ever, and we agreed that the nose was certainly not an important etiological factor. On one occasion he came to the office with one eye closed from an acute circumscribed œdema, and a number of similar œdematous patches on the face and scalp. They were not red, did not pit on pressure, nor did they burn or itch. On inquiry, he said he frequently had these outbreaks, usually on the head, and to which he paid no attention. I failed to appreciate at that time the probable relation of the asthma and the

skin lesions. It is probable that his asthma was due to an angioneurotic œdema of the bronchial mucous membrane, which had become more or less persistent.

As it affects the larynx, angioneurotic œdema becomes immediately a serious affection. It appears to come on during the night while the patient is asleep. Such was the case with two of my patients. It is entirely probable that many obscure and sudden deaths, occurring during the night, are of this nature, and an autopsy, unless this cause was looked for, would fail to find the cause. During the past few years cases are reported by Woodbury,[9] Hallock,[10] Leddermen,[11] Taylor,[12] Chittenden,[13] Ballenger[14] and others, in which a threatening dyspnœa from this cause occurred. Fatal cases are reported by Osler and Griffith.

Griffith's[15] case, in which both father and daughter died of œdema of the larynx from this disease, is reported in the *British Medical Journal*, June 14, 1902, and should be read in its entirety. He reports the case as "Hereditary Localized Œdema."

"The patient, a woman, was first seen by him in 1886, when she was eighteen years of age, because of dyspnœa, hoarseness, and dysphagia, and œdema of the arm and hand. The œdema involved the epiglottis and aryepiglottic folds, obscuring the view of the vocal cords. Tracheotomy was considered, but did not become necessary, and in a few hours the œdema disappeared. She gave a history of having had these transient œdemas, affecting all parts of the body, from birth. There was no periodicity in their occurrence. There was no history or evidence of any other disease, excepting that she was subject to bilious attacks.

"Her father, from his earliest infancy, had the same kind of swellings, and on three occasions they affected his throat: the first attack at the age of twenty-one years, the second a few years later, and the third at twenty-nine years, when he died as a result of it. In this last attack he came home from work, said his throat was swollen, and his physician was sent for. He came, but returned to his office for something to relieve him, but before his return the patient became suddenly cyanotic and died, asphyxiated.

"For sixteen years, from 1886 to 1902, Griffith saw his patient during many attacks affecting different parts of the skin and throat. The swellings were diffuse or circumscribed, usually pale, sometimes pink, did not pit on pressure, and were never bullous or vesicular. Twice the laryngeal dyspnœa was alarming. The tongue and buccal mucous membranes were at times the site of the œdema. The swelling came on suddenly, without warning, and almost as quickly disappeared. Finally, she had an attack, which proved fatal. At 10.30 A.M. she told her mother her throat was swollen, but that it was subsiding. At noon her husband came home, found her sitting on the floor amusing her child by spinning a top, and apparently free from dyspnœa. Suddenly she dropped the top,

tried but could not speak, clutched at her throat, got blue in the face, and died at once. Her mother, who was present at her death, said she died exactly as had her father before her. Griffith saw her a half-hour after death. The features were livid, tongue protruded, some fulness about the neck. The autopsy showed the larynx to be very œdematous, tense, and pale, and the sides of the laryngeal cavity in contact with each other. Transverse sections of the larynx showed that the œdema affected not only the mucous membrane, but the underlying deeper connective tissues and muscles. The œdematous fluid was serous, untinged with the coloring matter of the blood, and the tissues covering the true cords were decidedly affected."

The etiology of this peculiar disease is not at all clear. There are certainly three and perhaps four main etiological factors. Heredity is clearly shown in the classic case of Osler, reported in 1888, in which the disease appeared in five generations, and of the thirty-nine members of this family nineteen had angioneurotic œdema, two dying of œdema of the glottis. Griffith's case, just reported, is a striking one. All of my cases show the element of heredity. The peculiarity transmitted, Osler[5] says, must be either a morbid susceptibility of tissue or an inherited peculiarity of metabolism, or both combined. The individual attacks are probably provoked by some toxin produced within the body, as in the gastrointestinal tract, or in some other organ or tissue of the body, or it may be introduced from without. Gout and rheumatism are often mentioned as causes, but it is very doubtful if there is any connection. It is more probable that this disease is often mistaken for manifestations of gout and rheumatism. A certain poison or a certain kind of food may cause an attack in one person and not in another, or it may not produce a second attack in the same individual. My patients arrived at this conclusion in their own cases. Strawberries, fish, coffee, ice-cream, contact with a jelly fish are all given in the reports of cases as causes, but these cases are almost always simply the reports of single attacks. Exposure to cold and traumatism, worry, anxiety, mental excitement and fright, are doubtless all immediate causes for outbreaks. Antitoxin, as will be referred to later, is probably one of the various causes. These patients are invariably of a marked neurotic temperament. An interesting and an important question is as to the local condition determining, in a predisposed individual, the localization of the lesions, and Osler seems to think this is an important one in any individual case.

Collins[1] found that in 83 cases studied by him, the lesion first appeared in the larynx six times. He says the occurrence on any particular part of the skin or mucous membrane predisposes that part to other attacks.

There has been much question as to whether the colic, which occurs so frequently in the course of the disease, is the cause or the

result of the outbreak as it appears on the skin or in the throat. There is no doubt but that in mostly all cases the colic is the effect of the acute œdema localized in the stomach or intestinal tract. Morris[16] reports a case in which a portion of the gastric mucosa became detached in using the stomach-tube. It was examined histologically and showed an extreme œdema of the interstitial tissue, the lymph spaces and vessels being enormously dilated. The abdomen has been opened for supposed appendicitis when the condition was one of angioneurotic œdema of the intestinal mucous membrane.

The histological examination in Griffith's case of fatal œdema of the larynx showed that the œdema involved not only the mucous membrane, but the deeper connective tissue and underlying muscles. Crocker says that everything points to the disease being primarily a vasomotor disturbance, direct or reflex, central or peripheral; that a spasmodic contraction of the vessels is followed by a paralytic dilatation and stasis or retardation of the circulation in the papillary layer of the skin. Serous exudation then ensues, producing acute œdema. The same thing occurs in the mucous membranes and readily explains the sudden œdema and enormous swelling that comes on in a few seconds or minutes.

This same kind of œdema occurs in the brain and in the kidneys and probably in every organ or tissue of the body. Gruss[17] reports a case in which acute circumscribed œdema caused an acute retrobulbar œdema producing proptosis, and associated with cerebral symptoms. Osler's Case XV. had repeated attacks of aphasia and hemiplegia, the lesions on the skin being those of purpura, urticaria, and angioneurotic œdema during the fourteen years of the disease.

Ten days after the injection of antitoxin, given for prophylactic or therapeutic purposes, eruptions on the skin occur in a considerable percentage of the cases injected. These eruptions belong to the erythema group and are in order of their frequency, urticaria, erythema, purpura, angioneurotic œdema. The joints frequently become swollen, and there are evidences of acute swellings in the mucous membranes of the respiratory and gastrointestinal tracts.

Antitoxin from the same bottle may be injected into several children, and yet but one may have a skin eruption develop later, showing that the serum is not alone at fault, but that there exists in the affected patient a "peculiar susceptibility of tissue" or an inherited tendency toward one of the erythematous diseases; the antitoxin or something in it being simply the poison that provokes the attack in that particular person. Morse[18] reports a case in which, five days after a prophylactic dose of 500 units, the child complained of chilliness, prostration, vomiting, and œdema of the uvula and pharynx, the acute symptoms subsiding in thirty-six hours. Two years before this same child, after a prophylactic dose of 700 units, developed an urticaria of the skin.

It can readily be seen that when diphtheria exists in the throat, an acute œdema developing during the course of the disease would be attributed to the diphtheritic infection rather than to the antitoxin injected a few days before, and yet it might be due entirely to the latter. From our knowledge that angioneurotic œdema and urticaria occur in the throat, there is no reason to suppose that antitoxin causes these lesions in the skin and not in the fauces, pharynx, or larynx. There is every reason to suppose it does, and it is quite presumable that some of the suddenly fatal dyspnœas occurring in diphtheria are the result of the remedy operating on a peculiarly susceptible individual rather than to the disease diphtheria. The real cause of death in such cases would be overlooked, being masked by the disease. It is possible that the local lesions of diphtheria, as well as the toxæmia, might prevent the development in the throat of lesions which the antitoxin might, however, produce on the skin.

The cases which have attracted most attention have been those suddenly occurring deaths immediately following the injection of antitoxin, and usually in persons who did not have diphtheria, but were given prophylactic doses because of their exposure. The cause and effect here are not open to much question, but there has been much discussion as to how and why the antitoxin caused death.

It appears to the writer that in these cases the children have probably an inherited tendency to angioneurotic œdema, that the antitoxin has been the exciting cause of an attack, and that the swellings have occurred in the larynx or the lungs, producing a sudden œdema that causes asphyxiation, or the œdema may have occurred in the medulla or the brain, causing a paralysis of the respiratory or cardiac centres with resulting death.

For instance, Halderman[19] gave a prophylactic dose of antitoxin to a five-year-old child, who did not have diphtheria. It was asleep when the injection was given and almost immediately the lips began to puff up and in five minutes breathing ceased. Ninfong[20] gave a fifteen-year-old boy a prophylactic dose of 1500 units. Ten minutes later the boy became pale, extremities numb, cyanosis developed, the cheeks became swollen, and free vomiting occurred. Nitroglycerin was given and artificial respiration was resorted to, but the boy died thirty-five minutes from the time of injection. Prof. Langerhans'[21] son was given a prophylactic dose of antitoxin, and almost at once he began to vomit and soon died of suffocation. The autopsy revealed the trachea and bronchi filled with stomach contents and the uvula swollen. Adamkiewicz thought death was due to shock or pain resulting from the forcible injection under the skin of a large amount of fluid. Langerhans insisted that death was due to the poisonous action of the serum upon a healthy subject when unneutralized by a pre-existent toxin.

These cases would seem to have had an acute circumscribed œdema; in the first case of the lips and probably of the larynx;

in the second of the cheeks, the stomach, and the larynx; in the third of the uvula, and probably of the stomach; in each case the œdema coming on within a few minutes and causing death in from five to thirty-five minutes. None had diphtheria, all having received prophylactic doses.

Kortright[22] gave a sixteen-year-old girl 10 c.c. Behring's antitoxin, making the injection in the left side of the abdomen. She immediately said she "felt a lump in the stomach," and Kortright assumed she meant that she felt the swelling of the injected serum under the skin; but it is equally probable that what she felt was an acute swelling in the gastrointestinal tract. Five minutes later she complained of tingling and became restless, and immediately had a general convulsion. This was followed by general tonic and clonic convulsions, opisthotonos, cyanosis, and cessation of breathing. Artificial respiration was kept up till heart ceased in three minutes, death occurring in eight minutes from time of injection. An autopsy made eighteen hours later showed the brain and meninges, kidneys, and liver all markedly congested. No foreign body in larynx or trachea.

In Applegate's case, a boy, aged four years, having diphtheria, was given 15 c.c. antitoxin. One hour later he became very restless, vision completely lost, and three hours after injection died in convulsions.

Kortright was of the opinion that his case died because of something undiscoverable connected with the antitoxin or the injection. It is probable that had another child received the same injection nothing untoward would have followed—there being in his case the unfortunate combination of a predisposed patient and an exciting cause. In these 2 cases the localized œdemas were probably in the brain.

If these statements regarding antitoxin be true, it is not a reason or an argument against the use of this specific in the treatment of diphtheria, because it has saved thousands upon thousands of lives which otherwise would have been lost and has probably caused the death of but a very few. It is, however, an argument against the indiscriminate use of antitoxin for prophylactic purposes, because a single death from its administration in a person who has been simply exposed and had not contracted, and might not contract, the disease is frightfully deplorable. When it is used prophylactically, the previous personal and family history of the patient as regards the erythematous skin diseases should be inquired into, and if this develop the presence of or tendency to any of these diseases, then the antitoxin should be withheld until diphtheria actually occurs, or at least until a bacteriological examination of the throat in such an exposed person shows the Klebs-Loeffler bacillus.

Some makes of serum seem to have a larger percentage of skin eruptions follow than others, and we see much less of these erup-

tions now than three or four years ago, showing apparently that the present methods of preparing and "ripening" antitoxin is eliminating some of its deleterious properties, and it may be that in the future antitoxin may become inert as a cause in predisposed individuals, of the production of the erythematous lesions on the skin and mucous membranes.

The treatment of angioneurotic œdema as it affects the upper respiratory tract may be divided into the management of the various acute attacks as they arise, the correction of any local disease which may possibly determine the localization of the lesions or œdemas in the air passages, and the treatment of the constitutional disease back of the attacks. Of the last I have nothing to say beyond the fact that each individual case must be studied by itself, and if possible the underlying cause found and, if it can be, corrected. Osler has called particular attention to the fact that of his 29 cases of the erythema group, 24 per cent. finally died, 5 of the 7 deaths being due to uræmia. He has never seen, however, a case of pure angioneurotic œdema develop nephritis, and the fact is that cases in which angioneurotic œdema is the only lesion throughout the course of the disease are very few. For eighteen years my Case II. has apparently been such a case.

The treatment of the localized œdemas in the fauces, pharynx, and larynx would depend upon the severity. I have used both adrenalin and cocaine locally with apparent benefit, and in more severe cases, scarification of the pharyngeal and laryngeal œdemas. Should the dyspnœa increase and threaten life, tracheotomy should be done without hesitation or delay. Ice locally is probably contraindicated and might add to the œdema. Delbril advises brisk friction of the skin to induce an attack of urticaria on the skin when there is œdema of the larynx. Packard's cases of asthma would bear out the wisdom of this. Quiet in bed, reassurance, and, as far as possible, the removal of anxiety are important. These patients are highly neurotic, and fright alone might precipitate a sudden and fatal termination. Internally saline laxatives and alkalies to reduce the usual hyperacidity of the urine and increase the quantity are indicated. My patient, Case II., felt that he knew when an attack was coming on by the increased hyperacidity of his urine, and at once began to take alkalithia, which he thought lessened the frequency and mitigated the severity of his attacks, though it did not prevent them.

It would seem to be a sensible thing to treat and remove, if possible, between attacks any local disease that may exist in the throat or upper air passages, with a view to removing the localization of the acute circumscribed œdemas in these areas. It is doubtful if this would accomplish such a highly desirable result, but it certainly should be attempted.

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MILK AND SCARLATINA.

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MILK-BORNE epidemics of scarlet fever have been reported with more or less frequency ever since the English, during the latter half of the last century, called the attention of the medical profession to this method of spreading the scarlet-fever contagion. Such epidemics have been traced in some instances to the exposure of milk to infection from scarlet-fever patients, in others to milk from cows suffering from a disease supposedly analogous to scarlet fever in human beings. From the first the English have been the ones to lay the greatest stress on the agency of milk in carrying infection, as can be seen from the fact that of 330 epidemics of infectious disease, traced to contaminated milk, no less than 243 have been recorded by English authors (Kober).[1] The usual reason given for this preponderance of milk-borne epidemics in England is that Anglo-Saxons are the only people who drink raw milk to any extent; other European peoples habitually heat or boil their milk (Freeman,[2] Kober, Hall,[3] Jürgensen[4]). It is true that America has reported only 52 epidemics as against the 243 of England, but this is said to be due to laxness of sanitary supervision of the milk supply in this country, and lack of careful search for causes in the presence of epidemics (Freeman). As further proof of the agency of milk

in disseminating scarlet fever, many authors point to the fact that countries in which milk is not used as an article of diet, or very sparingly used by children, are free from scarlet fever. Thus the disease is practically unknown among the Japanese and very rare among the Hindoos.

In reading the literature on this subject one is impressed with the difference in the attitude of Continental and of British writers. While the former admit that apparently milk is a good culture medium for the virus of scarlet fever, and that there is evidence of infection having been conveyed from one human being to another through this agency, yet their attitude toward specific instances of such milk-borne epidemics is critical, if not skeptical. They insist that before charging the milk with the responsibility of carrying the contagion, other more usual methods must be ruled out, and there must be clear proof of the relation of the milk to the spread of the disease. The responsibility of milk in the spread of scarlet fever cannot be regarded as proved beyond reasonable doubt, unless the outbreak in question presents the following features:

The epidemic must be either distinctly localized or in scattered but sharply defined foci. It must be sudden in its origin and subsidence. The persons affected in the early days of the epidemic must be drinkers of milk from a certain supply. Attendance at the same school, play-grounds, Sunday-school, etc., must be excluded. It must be shown that the milk was exposed to the contagion of scarlet fever, and the epidemic should subside after the supply of infected milk is cut off. Direct sources of infection should be ruled out, as when disease is carried by the boy who delivers the milk, or when the buyers of the milk come in direct contact with the patient in the shop.

Very few reports have proof of this character, most of them failing to exclude sources of contagion other than the milk supply. Harrington[5] gives an interesting illustration of the erroneous conclusions which may be arrived at by insufficient probing of the evidence. In an outlying part of Boston, in 1897, a sudden epidemic of scarlet fever broke out among the children of well-to-do families. Nearly all the families took milk from the same dealer. No fault could be found with the dairy farm; the cows were healthy, and there was no scarlet fever among the dairy workers. The fact, however, that all of the cases appeared in families using the same milk pointed strongly to this as the source of the contagion. Had the inquiry stopped there this epidemic would have been added to the long list of milk-borne epidemics, but a more careful search revealed the fact that the first case of all appeared in a family which had milk from another source; that this child had played with and infected other children, who had spread the disease still farther. The children belonged to the same social set and were playmates. "Thus, what might have served as a most useful example of a milk-borne epidemic fell to the ground."

As an instance of a carefully studied and critically framed report may be quoted Tingvall's[6] account of an epidemic in Westeras, Sweden. During 1901 there were only 27 cases of scarlet fever throughout the whole city. In January, 1902, within four days, 27 cases developed in 22 families living in the same part of the city. No case of scarlet fever had occurred in this region in recent times. The stricken families had nothing in common except the source of their milk supply. Eighteen families bought milk from a dairy farm almost daily; three more bought it occasionally, and almost all drank it uncooked. The one remaining family had bought milk from a farm where there had been scarlet fever shortly before. Two cases of scarlet fever were discovered in the house where the employés of the dairy farm lived. After January only 11 cases of scarlet fever developed in the whole city during the rest of the year, and none in that part of the city.

Another instance, reported as early as 1870 by Oscar Home Bell,[7] leaves nothing to be desired in the way of evidence. He describes an outbreak of scarlet fever which appeared first at St. Andrew's University; then, after the university closed for the holidays, and the milk formerly supplied to it was distributed in the town, the disease appeared in the town. The wife of the dairy farmer had a sore throat and two of her children had typical scarlet fever. She nursed them, and, at the same time, milked the cows and handled the milk. The households in which the cases occurred were, with one exception, houses supplied from this farm, and all the households taking the milk had scarlet fever except one, in which there were no children. Two boys, who had already had scarlet fever, called at the farm regularly for milk and carried it home, as a result of which three other children in the family, who had not visited at the farm, took scarlet fever.

Another careful report is that of the Rostock epidemic given by Jürgensen,[4] who is able to state that those drinking the milk boiled escaped infection. Certain of those cited by Freeman[3] are also satisfactory, as are some in Kober's[1] list, many in Hart's[8] first tabulation, some in his second,[9] Wende's[10] account of the recent Buffalo epidemic, and Eckholm's[11] account of an epidemic in Wasa.

On the other hand, the English journals are full of reports which ignore ordinary rules of evidence, and which incline one to suspect that the theory of the agency of milk in disseminating scarlet fever has taken so strong a hold upon that country that, in the absence of direct proof to the contrary, an English health officer is apt to assume that any localized epidemic of this disease is traceable to contaminated milk. The pages of the *Lancet*, of the *British Medical Journal*, and of *Public Health* abound in such reports as the following: "An outbreak of scarlet fever in Clapton. Dr. J. King Warry was able to associate the outbreak with the distribution of a particular milk supply, but he could find no evidence as to the manner

in which the milk became infected. There was no reason for thinking that the milk was infected from a human source."[13]

"Mr. Henry Armstrong has a careful report of an outbreak of scarlatina, apparently associated with the use of milk from a certain dairy. Of the 19 cases 17 took milk from the same dairy. There was some suspicion of scarlet fever in the family of a dairy worker some few months before the outbreak, but there were no other cases among the 219 other families taking the same milk."[13]

"Dr. Wallace regards 8 out of 26 cases in October as clearly connected with this contaminated milk, and 4 out of 21 cases in November as unquestionably due to the same cause." This cause was "unrecognized cases of scarlet fever in the person of the milk dealer and his child," who had suffered from "cold and sore throat." The cases were not treated medically.[14]

"In one street 21 families (more than half those supplied with the milk) yielded 29 cases; some streets taking the milk suffered no attacks. The active principle of contagion was therefore apparently not evenly distributed."[15] "Exciting cause of epidemic: Illness, suggestive of scarlatina, in the families of the two milkmen supplying the implicated milk, upward of three months prior to the outbreak."[16] "Epidemic sudden and cessation well marked; of the 205 households invaded, 74, or 34 per cent., were supplied with milk from one farm. Exciting cause of outbreak not ascertained."[17] "Scarlatina, which had hitherto chiefly affected the poorer quarters, suddenly blazed out in better-class houses exclusively, and three-quarters of the homes invaded dealt with one dairy. Cows in a febrile condition, due doubtless to the unsanitary character of their housing accommodations. A sample of mixed milk of several cows showed bacteria (character not given)."[18]

In strong contrast to this attitude are the unenthusiastic utterances of German and of some American writers:

"Observations have recently been made which indicate that it (the scarlet-fever contagion) may be conveyed in milk" (Osler[19]). "The possibility is not excluded that scarlatina can be transmitted by means of the milk" (Jürgensen[4]). "In England it has been thought that the contagion may be carried by milk" (Strümpell[20]). "Without examining into the trustworthiness of the published reports of epidemics of scarlet fever attributed to the milk supply, I would simply state that it is extremely difficult to produce indubitable proof of such occurrences so long as we are in ignorance of the specific cause of the disease. However, the mere possibility that so malignant a disease may be communicated in this way deserves our full attention" (Eckholm[11]). "The question should not be regarded as definitely disposed of, but is one that merits the most searching investigation by pathologists and bacteriologists" (Kober[21]).

This difference between English observers and those of other countries cannot be explained simply on the ground that milk-

borne epidemics of scarlet fever are more common in England than in other countries. Undoubtedly, the fact that the English drink their milk unboiled explains why there should be a greater number of milk-borne epidemics of infectious diseases there than in Germany, France, Austria, or Italy, but does not explain the enormous difference between their record and that of the Scandinavians, who also use unboiled milk. Kober[1] gives the list for Scandinavia as 11, as against the 243 of England, and Eckholm[11] speaks of the milk-borne epidemic of scarlet fever at Wasa, in 1903, as only the third thus far reported in Sweden.

In all the epidemics thus far considered the contagion is supposed to have passed from a case of human scarlet fever to the milk, and to have been carried by the milk to a healthy individual, who thereupon contracted the disease. Admitting this to be not only a possible but a fairly frequent mode of infection in scarlet fever, what shall we say as to that second class of milk-borne epidemics—those, namely, in which the disease is supposed to originate in the cow, and the contagious virus to be present in the milk at the time of milking?

The English are practically the only adherents of the theory that scarlet fever is a disease of cattle and can be conveyed from them to human beings. Their literature is full of references to outbreaks of scarlet fever and diphtheria originating in the milk of diseased cows. The first epidemic attributed to "what we now confidently recognize as scarlatina in the cow" (Hart[9]) broke out in 1885, and an investigation, ordered by the London Local Government, was carried out by W. H. Power, Klein, Cameron, and Wynter-Blythe.[22] As a result, the disease was found to be limited to the users of the milk from a dairy farm at Hendon. W. H. Power was convinced that the milk drunk by these scarlet-fever patients came from certain cows which were suffering from a vesicular and ulcerative eruption of the udder. This eruption was directly communicable from the cows to the hands of the milkers, and from them again to other cows. Klein isolated from the vesicles of both cows and milkers a streptococcus which, when injected into calves, produced in them visceral lesions resembling those of scarlet fever in man, namely, congestion of the lungs, parenchymatous degeneration and congestion of the kidneys, and hemorrhages in the spleen. He also isolated the streptococcus from the milk of the diseased cows, and from the blood of 4 out of 11 cases of scarlet fever, which streptococci he used to produce a local vesicular eruption in calves. Therefore, he concluded that this organism is the cause of scarlet fever in man, and of this particular ulcerative eruption in cows. Cameron gave a detailed description of the disease among the Hendon cows, calling it a specific, contagious, constitutional disease, capable of being communicated to man. A milker received the virus into a recent scratch on the forefinger. General weakness, malaise, loss

of appetite resulted, and, after about four or five days, a vesicle or small blister appeared on the finger; later, several on the back of the hand. The whole hand became swollen and inflamed, the inflammation extending in broad lines as far as the elbow. The general disturbance lasted a fortnight. Cameron considered the disease quite distinct from cowpox. Calves inoculated with scrapings from the ulcers developed vesicles at the site of inoculation.

The report of Power, Klein, Cameron, and Wynter-Blythe aroused much comment, and from this moment we find the attention of British physicians turned to the now famous "Hendon cow disease" as a frequent cause of epidemics of scarlet fever in man. The literature of epidemics originating in the use of milk from diseased cows increases rapidly, and it soon becomes unnecessary to show that the disease in question has the characteristics of the "Hendon cow disease." It is sufficient to state that the cows were suffering from an "obscure febrile condition;"[23] that they were "off their feed, giving ropy milk, and had scales on the teats"[24] or "abraded teats,"[25] or a rash over the body consisting of "small, red pimples."[26] Excrementitious matter or vaginal discharge may also cause contamination of the milk by the virus of scarlet fever. Thus, Power, quoted by Hart,[9] finds the origin of certain cases of scarlet fever in the use of milk from a cow who had "lost portions of her coat," and whose "buttocks and posterior udder were fouled by excrementitious matter and, perhaps, by vaginal discharge as well." Sewage-polluted water drunk by the cows, or used to wash milk-cans, is also supposed to be responsible for scarlet fever in the drinkers of the milk. Scarlyn Wilson[27] considers the discovery of "bacteria" in milk as confirmatory proof that the milk of cows kept in filthy surroundings caused scarlet fever in children, and the *British Medical Journal* makes respectful editorial comment on his report. The enthusiasm for this theory of the direct causative relation between a diseased condition in cows and scarlet fever in drinkers of their milk becomes so great that, in 1897, we find W. H. Power and Ernest Hart[9] not only accepting all the reports up to date, but adding many still more wonderful. Thus, Hart discusses the question whether a certain epidemic of scarlet fever was caused by the rinsing of milk-cans in sewage-contaminated water or by the use of milk from a cow newly calved, and inclines to the latter belief. The fact that the milk of newly calved cows can cause scarlet fever is stated by Hart to be "an important discovery of modern times," due largely to the researches of Power. Apparently the same kind of milk can cause different diseases. Thus, "ropy milk" caused an outbreak of diphtheria in Hendon in 1883, of scarlatina in Charlton in 1892, and of typhoid fever at Warminster in 1895. It is distinctly stated by Hart that the disease among the cows at Glasgow, which was supposed by Russell to be responsible for an outbreak of diphtheria in that place, was the same as the "Camberwell cow malady,"

which was considered by Sir George Buchanan and by Klein to be the cause of an epidemic of scarlet fever. The milk of newly calved cows caused scarlatina in London in 1883, at Macclesfield in 1889, and at Sutton Coldfield in 1891; while at York Town and Camberley in 1886 the same sort of milk caused diphtheria, and in Dundee in 1889 it was the "vera causa of infective quality of milk causing typhoid fever." Ernest Hart cites all these reports and concludes: "Thus, we are face to face with a fact, and it is the more important when we remember that the condition attaching to newly calved cows is not one which has been deemed of any importance by veterinary inspectors. Indeed, we may be pardoned for saying that the subject of milk-borne disease is furnishing a history of long struggle by medical as opposed to veterinary opinion. But this struggle has been harder in the matter of eruptive diseases of cows than on any other point, and the victory has certainly been with the medical experts."

Nor are these extreme views confined to one or two writers. In 1896 we find Thorne-Thorne[28] stating that the milk from diseased cows can communicate scarlet fever to man. Editorials in the *Lancet*, as late as 1892, add their testimony to the same effect, and so do the editorials in the *British Medical Journal*, as late as 1894.

Not all of the British authors, however, and practically no one in countries outside of England, hold the views outlined above. (See Allbutt's *System*, vol. iii. p. 163.) At the time of the government investigation of the famous Marylebone epidemic there were voices raised in dissent to the conclusions reached by Klein and Power, but the dissenters, being chiefly veterinarians, did not command much respect from the medical profession. The Agricultural Department of the Privy Council was roused by the statements of Power and his colleagues to make a searching inquiry into the disease among the Hendon cows, which was said to have caused scarlet fever in man. They entrusted the inquiry to Edgar Crookshank,[29] whose report is careful and convincing, and shows a singularly modern attitude toward the bacteriological aspect of the question. Crookshank had an opportunity to investigate an epidemic among cows which occurred in another county, but which was apparently the same as that just previously seen at Hendon, corresponding in every respect to the description of the latter and being communicable to calves and to milkers by direct inoculation. From the lesions on the cows' udders and on the hands of the milkers he isolated Klein's streptococcus. But among the 1500 to 2000 drinkers of the milk from this herd there was not one case of scarlatina, and neither here nor at Hendon did the disease which the milkers contracted resemble scarlet fever. He studied the lesions on the hands of the milkers and concluded that they were those of "typical Jennerian cowpox," and that the irritation and abrasion in the process of milking was responsible for the

difference in appearance of the ulcers on the cows from normal pocks. Lymph taken from the human vesicles caused typical vaccinia in calves.

Crookshank concluded that Klein's streptococcus is identical with that found in diphtheria, erysipelas, puerperal fever, scarlatina, etc., and that when inoculated into animals it sets up simply a septic inflammation, not a specific infectious disease. The lesions produced experimentally by Klein pointed only to sepsis, not to scarlatina. When this streptococcus is found in the blood of scarlet-fever patients it should be regarded as a complication, not as causally related to the scarlet fever. Other veterinarians, as Brown and MacFadyen, concurred in this report. Brown[30] stated that a disease, identical in the opinion of veterinarians with that at Hendon, prevailed in parts of eleven counties in which no scarlet fever was found. The outbreak at Hendon was, therefore, not an isolated one, and was elsewhere not associated with scarlet fever in man.

In Germany and in America the general opinion held is that the "Hendon cow disease" was cowpox, or something closely allied to it, and that the scarlet-fever epidemic was only coincident, or that it was not true scarlet fever, but a septic fever due to the presence of pus in the milk. Careful examination of the reports, unfortunately often very meagre, of cases of scarlet fever caused by the drinking of milk from diseased cows brings to light certain features which suggest a disease other than typical scarlatina. Baginsky,[31] for instance, comments on the mild character of the disease in the Hendon epidemic, only 1 fatal case among 60, and the short incubation period of two days. The statement as to a one to two-day incubation period is also made by C. N. Cooper.[32] Clothier,[33] in his account of the outbreak at Stroud Green, says: "As has been observed in previous epidemics of scarlatina disseminated by milk, the type of the disease is mild, and no deaths are reported."

Harrington[34] abstracts the report of an epidemic in Lincoln, England, in 1903, involving 40 persons, the majority of whom were seized on the same day and none of whom was seriously ill. There was a remarkable susceptibility to infection in this epidemic, no less than 14 persons in one household suffering from the disease, a fact which certainly would in itself incline one to doubt the diagnosis of scarlet fever. The milk to which this attack was attributed came from cows with vesicles on the udders. In an epidemic recorded by G. Buchanan[35] the persons attacked with what was held to be scarlet fever, 19 in number, were all adults. Cream of which all had partaken was held responsible for the outbreak, but there was no evidence that any case of scarlet fever had come in contact with it.

Now, if we assume that the milk in these instances was infected, not with the specific virus of scarlatina, but with pyogenic cocci from the inflamed udders of the cows, the matter becomes clear at once. That mammitis in the cow is accompanied by the appear-

ance of streptococci and staphylococci in the milk has been proved by many investigators. Guillebeau[36] examined the milk of 76 cows with inflammation of the udder and found that all contained pyogenic cocci virulent for animals. Nocard and Mollerau[37] demonstrated streptococci in the milk of cows with mammitis, as did also Adametz,[38] Hueppe[39] and others, to whose researches must be added those of Klein, cited above.[29] Bergey[40] found that streptococci and staphylococci persist in the milk for some time after all external signs of mammitis have subsided. At the height of the inflammation these germs are present in enormous numbers. The descriptions of the condition of the cows in the Hendon epidemic, and in others resembling it, show that pus, scales, and blood must have found their way into the milk through the friction of the milkers' hands. Dr. Russell[41] found cocci in the milk of a cow suffering from the Hendon disease, and also in the blood of the calf she was suckling. In other cases it is apparent that the cows were suffering from a catarrhal mammitis at the time, or shortly before the epidemic among drinkers of the milk. The theory of Power as to the infectious nature of the milk of newly calved cows may have some foundation in the greater tendency to mammitis just after calving, or possibly the cows may have been suffering from a mild form of puerperal sepsis.

There are, in fact, two or three reports of cases of throat inflammations attended with constitutional symptoms, which apparently came from the drinking of milk containing pus. Kober[21] describes two epidemics of follicular tonsillitis in a boys' school, with severe gastric disturbances; one involving 50 and the other 84 cases. The cows showed the lesions of variola vaccinia. In 1869 Fagan[42] reported a case of pseudomembranous stomatitis following the drinking of milk contaminated with pus and blood from an abscess in the udder of the cow. Gray Edwards[43] describes with much care and accuracy some cases of follicular tonsillitis due to infected milk. Cultures from the throats of the 12 cases and from the milk yielded the same organisms, namely, staphylococcus aureus and albus and streptococcus. Three children who drank only boiled milk escaped the infection. The "ropy," or "stringy," or "slimy" condition of the milk mentioned by some authors, and especially emphasized by Power and Klein as the cause of scarlet fever and diphtheria, is a condition caused by a wide variety of organisms, including certain strains of staphylococcus pyogenes (Passet, Lehman, and Neumann), and various other cocci and bacilli.*

* Bac. lactis sapon. (Weigmann and Zirn, Centralbl. f. Bakt., Bd. xv. p. 463); Micrococcus Freudenreichii and Bacterium Hessii (Guillebeau, Centralbl. f. Bakt., 1892, Bd. xi. p. 439); Bacillus lactis viscosus (Adametz, Centralbl. f. Bakt., 1891, Bd. ix. p. 698); Leischmann's bacillus (Centralbl. f. Bakt., Bd. xvi. p. 122). Reference to still others is made in Adametz's original article (Berliner Landw. Jahrb., 1891), cited above. Guillebeau's micrococcus Freudenreichii is considered by Lehmann and Neumann as probably a streptococcus. Neither his nor Adametz's organisms were pathogenic.

It is evident that milk-borne epidemics of scarlet fever do occur, but there seems no reasonable basis for the belief that they can originate in a diseased condition of the cow. In this connection we cannot do better than quote the words of Kober.[21] "When we consider that toxins may and do produce a scarlatinous exanthema, we feel warranted in saying that in all the epidemics of scarlet fever and diphtheria which were traced to milk from cows suffering from inflammatory lesions of the udder or from puerperal fever we have typical instances of a streptococcus and staphylococcus infection. . . . There is no proof that there is a disease in the cow which is communicable as scarlet fever to man."

In view of our ignorance as to the infectious agent in scarlet fever, it is interesting to know that milk serves as a good culture medium for it. Most authentic reports of this kind show that the milk was directly exposed to the contagion, as when the milking or handling of the milk or washing of the cows was carried on by persons suffering from a mild form of scarlet fever, or in the stage of desquamation following it. In many instances, however, the contact is less direct; a healthy person apparently carries the infection from a case of scarlet fever to the milk. Wives and daughters of dairymen nurse cases of scarlet fever and handle the milk at the same time. Some of these reports must undoubtedly be taken with caution, for the possibility is not excluded that the selling of the milk was carried on in the room where the sick person lay, or the delivery made by a person ill with scarlet fever, but in others the evidence of indirect infection is very clear. Of Hart's 99 epidemics of milk-borne scarlet fever, the disease was said to exist either at the dairy or on the milk farm in 68. In 17 of these, persons actually suffering from or recovering from scarlet fever handled the milk, but in others the connection was less direct. Two were supposedly caused by bottles or cans left in infected houses; 1 by the wiping of cans with an infected cloth; while in 3 the milk, though not handled by the patient, was stored in or close to the sick-room. In 6 instances dairy employés lodged in or had visited infected houses. Whittaker[44] gives an instance of infection of the milk and of the milkman's daughter through rags stored in the house and used to wipe the cans. The girl came down with the disease in another town on the day following her departure from home.

Many authorities on scarlet fever treat with a certain suspicion the instances given in the literature of infection through a third person, himself free from the disease. As the scarlet-fever virus is not highly infectious, and is apparently confined to the air immediately around the patient, many are inclined to believe that the carrying of the infection by third persons is very rare. But if, as seems very probable, milk is a good culture medium for this virus, it is easy to see why it should figure prominently in such indirect

infection. Kerschensteiner[45] says that if infection is to take place through a healthy third person the "scarlet fever dust" must be blown to that person, must cling to his skin, hair, or clothes, and must be blown off from him to the third person; and as scarlet fever is not very infectious, a large amount of the infectious stuff is needed. Now, if we imagine the virus not carried in a dry condition and inhaled as dust, but planted in a liquid culture medium and then, after a lapse of time sufficient for its growth, brought in contact with the throat in the process of swallowing, it is easy to believe that infection through the medium of a healthy third person can take place with comparative frequency, and also that utensils exposed to the air of the sick-room, even at a distance from the patient, may become infected sufficiently to carry the disease.

CONCLUSIONS. The literature of milk-borne epidemics of scarlet fever contains many which are (*a*) based on insufficient evidence as to the agency of milk in the dissemination of the disease; (*b*) based on the erroneous belief that a disease in cows is capable of causing scarlet fever in man.

The disease in cows supposedly responsible for scarlet fever in man is ordinary cowpox, and the disease in man supposed to be caused by it is either septic fever from infection from pyogenic organisms or coincident scarlet fever.

There is no good evidence that milk from diseased cows can cause scarlet fever in man.

After rejecting many reports of milk-borne outbreaks of scarlatina there remains a fair number which are above criticism, and which apparently prove that milk is a good culture medium for the scarlet fever virus, and is the most frequent agent of indirect infection in this disease.

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THE CAUSE OF PULSATIONS IN EMPYEMA.

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Guilliam de Baillon in the sixteenth century reported the first authentic case of pulsating empyema.

Le Roy (1776) speaks of the possibility of pulsating empyema in his Aphorisms, 489: "When the pus sac lies on the heart and large vessels in such a manner as to transmit their movements there develop false impressions of an aneurysm."

Berard (1836), Stokes (1838, first used the term pulsating empyema), MacDonnell (1844, first used the term empyema necessitatis pulsans), Walshe (1851), Wintrich (1854), Flint (1856), Aran Heyfelder, Owen Ress (1858), Schoostek (1865), Gourbon (1870), and Muller Plagge, Traube (1872), Frentzel (1875), Topham and Dunin (1878) each report one case.

Traube thinks the pulsations are due to a pericardial effusion associated with the empyema.

Comby (1881), reviewing the literature, reported a case and explained the pulsation by saying: "When the lung is completely collapsed, pushed to one side and adherent to the pericardium, it no longer resists the heart, the pulsations of which are then transmitted to the fluid in the pleural cavity, thence to the thoracic wall."

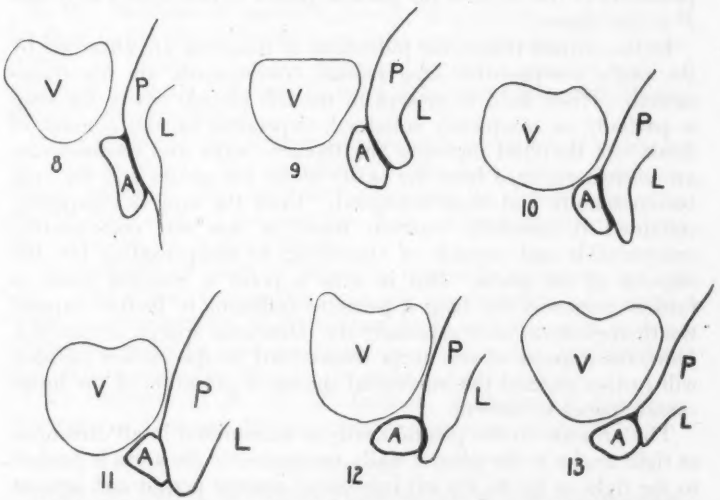
Fereol (1884) reported a case of pyopneumothorax pulsans and says the pulsations are due to the presence of the air.

Skoda Gutbrod says, "In systole the heart has a right to left movement which may explain the pulsation."

Frantzel contradicts Traube's theory that a pericardial exudate is necessary to cause pulsations.

Kussmal says, "When the pleural wall next the heart is thick enough to resist the impulse of the heart, the heart beat is transmitted through the pleural wall to the fluid."

The recent literature, at my disposal, adds no new theories to account for the pulsations, and in each the author states that the theories advanced are inadequate.



The relationship of the aorta to the parietal pleura in six transverse sections of the thorax is shown. Sections of the trunk are numbers eight, nine, ten, eleven, twelve, and thirteen. *V*, vertebra; *A*, aorta; *P*, pleural cavity; *L*, lung. Section eight is on a level with the upper border of the sixth vertebra; nine, with seventh vertebra; ten, with eighth vertebra; eleven, with ninth vertebra; twelve, with tenth vertebra; and thirteen with eleventh vertebra. From the point where the aorta touches the vertebra until it enters the diaphragm it is in immediate contact with the parietal pleura.

At the level of sections number nine, ten, eleven, twelve, and thirteen the right parietal pleura could easily be forced against the aorta.

While working on cross-sections of a body with pleurisy of the left side, the anatomical relationship of the organs suggested to me an explanation for the pulsating empyema which on analysis seems to fulfil all the requirements.

The requirements are a firmly fixed pulsating organ; contact of the pleural wall with this pulsating organ; distention of the pleural sac with fluid, air, or solid material, and collapsed condition

of the lung. The first requirement is fulfilled by the thoracic aorta; the second, by the normal relationship of pleural wall to thoracic aorta; the third, by presence of fluid, pus, or a combination of these with air in the pleural cavity; and the fourth, by the collapsed condition of the lung in pleurisy and empyema.

The thoracic aorta above lies slightly to the left of the median line on the bodies of the vertebra. In the lower part of the thorax it is almost in the median line. Immediately over the walls of the aorta is the parietal pleura. This relationship continues from the fourth to fifth rib above as far down as the diaphragm. The aorta is firmly anchored by the intercostal arteries, so that it requires considerable force to move it either to the right or left. This proximity of the aorta to the parietal pleura is well shown at *A* and *P* in the figure.

In the normal thorax the pulsations of the aorta are absorbed by the easily compressible lung tissues, consequently are not transmitted. When fluid is present in the left pleural cavity the lung is partially or completely collapsed, depending on the amount of fluid. As the fluid increases the thoracic walls and mediastinum are pushed outward from the cavity of the left pleura and the lung becomes more and more collapsed. Until the lung is completely collapsed it naturally contains more or less air, consequently compressible and capable of absorbing, or compensating for, the impulse of the aorta. But in time a point is reached when to further compress the lung a pressure sufficient to further expand the thoracic wall, more especially the intercostal spaces, is required. Now, the impulse of the aorta transmitted to the pleural effusion will further expand the intercostal spaces or pulsation of the intercostal spaces is present.

The pressure in the pleural cavity is transmitted in all directions at right angles to the pleural walls, consequently the aorta is pushed to the right as far as the left intercostal arteries permit and against the vertebral column. The arterial wall expands in all directions. The artery being firmly fixed against the vertebral column and nearly half-covered by the parietal pleura, which does not offer a resistance equal to that offered by the vertebral column and stretched left intercostal arteries, the aortic pulsations must be transmitted to the fluid in the pleural cavity. This pulsation of the effusion is transmitted equally in all directions and must be taken up by the elasticity of the mediastinal membranes, diaphragm, and thoracic wall and the compressibility of the lung. Naturally, of these structures the one with the greatest elasticity will absorb the greatest portion of the impulse. Normally, the lung is sufficiently elastic, consequently sufficiently compressible, to absorb the entire impulse; but when collapsed and solid its elasticity is much less then that of the thoracic wall, which must pulsate synchronously

with the aorta, or systolic in time. If the thoracic wall is weaker than normal, due to inflammation and degeneration of the muscles and parietal pleura or to paralysis of the intercostal muscles, the pulsations naturally will occur under a lower intrapleural pressure than when the thoracic wall is normal; or pulsation of the thoracic wall will occur in pleural effusions whenever the elasticity of the lung becomes less than that of the thoracic wall—in other words, when it requires more pressure to further compress the lung than it requires to further expand the thoracic wall. The mediastinal membranes and diaphragm probably pulsate to some extent. But the pulsation of the one is absorbed by the normal lung, and of the other, by the abdominal organs.

As this pulsation is purely hydraulic in principle, the nature of the fluid in the pleural cavity is of little or no importance. Consequently in pleural effusions pulsation is not a positive sign of empyema. In the literature at hand only three cases of pulsating pleurisy are recorded.

When a part of the fluid is withdrawn a cessation of pulsation has been recorded. The withdrawal of the fluid reduces the intrapleural pressure. The lung expands when its elasticity becomes greater than that of the thoracic wall. Consequently the lung again absorbs the aortic pulsation.

The majority of cases of pulsating empyema occur on the left side. Only a few cases in the right side are recorded. An examination of cross-sections of the thorax shows that the right parietal pleura may easily be pushed against the aorta so that its pulsations may be transmitted to fluid in the right pleural cavity.

I am indebted to Dr. C. M. Jackson, Professor of Anatomy, University of Missouri, for the transverse sections of the trunk.

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AN INVESTIGATION OF THE INFLUENCE OF ADRENALIN CHLORIDE ON TOXIC DOSES OF COCAINE.[1]

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THE danger associated with the use of general narcosis has led to the employment of numerous drugs and methods of admin-

[1] Read at the Annual Meeting of the Second District Branch of the New York State Medical Association, held at Saratoga in May, 1905.

istration in the hope of finding a perfect local anæsthetic. On this account, the introduction of cocaine by Koller met with ready acceptance; and the discovery of spinal anæsthesia in 1884 by Corning (rediscovered and extensively used by Bier) gave an additional stimulus to the use of cocaine.

The great disadvantage associated with the use of a local anæsthetic is the danger to the individual resulting from the absorption of the drug used as the anæsthetic. Cocaine is such a drug. It is almost perfect in its local application, making possible the severest operations free from pain, and yet at times cocaine poisoning may result from the absorption of the drug. Various methods have been employed in the endeavor to prevent the occurrence of this toxic action. Since the toxicity of cocaine presumably comes from its entrance into the blood the first need was a knowledge of a means of hindering its absorption.

One of the first methods used to accomplish this was that of Reclus and Schleich, with the introduction of what is known as Schleich's solution. The anæsthesia depends upon the bulk of the fluid. Large quantities of a weak solution of cocaine are injected and thus only a small amount of cocaine comes into contact with the tissue, and the danger of absorption is thus minimized. The chief objection to the use of Schleich's solution is the œdema produced and the consequent loss of normal appearance of the tissue. The constriction of the circulation above the part to be anæsthetized, in order to prevent absorption, has marked limitations. The use of an ethyl chloride spray to cool the part previous to the injection of the cocaine, as introduced by Braun, has not been altogether satisfactory. He has demonstrated that, so long as the vitality of tissue is kept lowered by cooling, absorption is hindered.

Klapp experimented on dogs with an oily solution of cocaine. He claims that an oily solution of cocaine prevents the capillaries and lymphatics from taking up the cocaine as rapidly as they otherwise would and by this means it prevents the toxic symptoms resulting from the absorption. Glycerin is found to have a similar action in retarding absorption.

The latest and most universal method for the reduction of the toxicity of cocaine is its use in combination with adrenalin chloride. Scarcely a medical journal can be perused without finding some reference to this new anæsthetic.

In the *Archiv f. klin. Chir.*, Bd. lxix., Braun gave the results of his experiments with combinations of adrenalin and cocaine. He employed rabbits for his experiments, giving subcutaneous injections of adrenalin chloride followed ten minutes later by similar injections of cocaine. Previous determination had shown that 0.1 gm. cocaine per 1000 gm. of body weight was a fatal dose. Two typical experiments are here given:

1. Rabbit weighing 1300 gm. injected subcutaneously with 1 c.c. of a 1:1000 adrenalin chloride solution. Ten minutes later 0.13 gm. cocaine injected subcutaneously into the same place. In five minutes paralysis, no convulsions. Animal recovered.

2. Rabbit weighing 1700 gm. injected subcutaneously in the head with 1 c.c. normal salt solution and with 1 c.c. of 1 : 1000 adrenalin chloride subcutaneously in the back. Ten minutes later 0.17 gm. cocaine injected subcutaneously in the head in the same place as the salt solution. In five minutes paralysis, convulsions. Animal recovered.

From these and similar experiments the conclusion was reached that previous injections of adrenalin inhibit the toxic action of cocaine. The inhibitory action also takes place, but to a less degree, when the injection of the two drugs is not made in the same place.

In a subsequent article (*Centralblatt f. Chir.*, Bd. xxx., No. 38), Braun confirmed his previous assertion as to the value of combining adrenalin and cocaine. In a third paper (*Archiv f. klin. Chir.*, Bd. lxxi.) he stated that the adrenalin cocaine mixture as compared to cocaine from the standpoint of an anæsthetic is quicker, stronger, lasts longer, and is less toxic.

Foisy in using a mixture of cocaine and adrenalin as compared to cocaine alone found:

1. Greater anæsthetic effect.
2. Greater local vasoconstrictor effect.
3. Less toxicity.

He mentioned two sets of experiments. The first set of experiments was on man with a mixture as follows:

Chlorohydrate of cocaine 1 : 200	10 c.c.
Adrenalin chloride 1 : 1000	10 drops.

This mixture was employed in 149 diseases in doses from 2 to 10 c.c. without experiencing the accident of cocaine intoxication. In giving the same doses of cocaine without adrenalin toxic effects, such as paleness, sweating, talkativeness, muscular tremor, and delirium frequently developed.

The second set of experiments was with subcutaneous injections in animals. The toxic dose of cocaine according to Laborde is 0.01 gm. per 1000 gm. body weight. With mixtures of adrenalin and cocaine in this dosage Foisy has never produced contractions or convulsions in animals.

Donitz reported a large number of experiments confirming Braun's findings. Cats were injected by subdural lumbar puncture and it was found that by injecting adrenalin and cocaine together the toxicity of the latter could be reduced one-third while injections of the adrenalin six minutes before the cocaine reduced the toxicity one-fifth.

Braun's findings were that: (1) adrenalin lessens the toxicity of cocaine; (2) it enhances its anæsthetic strength for duration, extent,

and intensity. Donitz finds that both these hold for subdural application.

The experiments of Ziegan as regards the amount of adrenalin chloride that can be injected without harm are of interest. He found that 0.001 gm. of adrenalin injected subdurally by lumbar puncture in cats caused anæsthesia of the hinder parts in eight to twelve minutes. If, after the injection, the animal was held with its head downward there resulted a general anæsthesia without loss of reflexes lasting thirty to fifty minutes. In two hours all effects of the drug disappeared. An injection of 0.003 gm. caused tonic convulsions in the lower extremity, and injections of 0.005 gm. killed by œdema of the lungs and paralysis of the respiration.

An interesting series of experiments was performed by Thies, the results of which were contradictory to those of Braun and Donitz. Cats were used for the experiments, nearly all the injections being made by lumbar puncture. The dosage and results are shown in the two following tables. Table A represents those cases where the adrenalin and cocaine were injected together. Each experiment has a control represented by an animal of equal weight receiving the same dose of cocaine without adrenalin admixture.

TABLE A.

0.02 C + 0.0005 A	Lived.	0.02 C	Lived.
0.02 C + 0.0005 A	"	0.02 C	"
0.02 C + 0.0005 A	Died.	0.02 C	"
0.03 C + 0.0005 A	Lived.	0.03 C	"
0.04 C + 0.0005 A	"	0.04 C	"
0.04 C + 0.0005 A	Died.	0.04 C	"
0.05 C + 0.0005 A	"	0.05 C	"
0.05 C + 0.0005 A	Lived.	0.05 C	"
0.06 C + 0.0005 A	Died.	0.06 C	"
0.08 C + 0.0005 A	"	0.08 C	Died.
0.1 C + 0.0005 A	"	0.1 C	"
0.1 C + 0.0005 A	"	0.1 C	"
0.2 C + 0.0015 A	"	0.2 C	Lived.
0.2 C + 0.001 A	"	0.2 C	"
0.25 C + 0.0005 A	"	0.25 C	"

From the above table it will be seen that the amount of cocaine necessary for fatal doses was greater where cocaine was used alone than in those cases where it was used in combination with adrenalin.

Table B shows the results where the adrenalin was injected by lumbar puncture followed in six minutes by the injection of cocaine. There are three of these experiments with controls of animals of equal weight receiving the same dose of cocaine.

TABLE B.

0.06 C + 0.0005 A	Died.	0.06 C + salt sol.	Lived.
0.08 C + 0.0005 A	"	0.08 C + salt sol.	Died.
0.08 C + 0.0005 A	"	0.08 C + salt sol.	"

Table B shows the same results as Table A. A certain dose of cocaine (0.06 gm.) injected in combination with adrenalin is fatal,

but the same dose of cocaine when used alone is not fatal. A dose of 0.08 gm. of cocaine causes death whether injected alone or in combination with adrenalin.

Thies observed that the symptoms of cocaine poisoning did not come on so rapidly in those cases where adrenalin was used. He concluded that the toxic action of cocaine was not destroyed or diminished by adrenalin, but was simply retarded. With fatal doses of cocaine adrenalin has therefore no effect in diminishing its toxic action.

In the *Berl. Klinik* for January 1, 1904, Braun gave a summary of the work done on adrenalin cocaine anæsthesia and discussed Thies' article. His criticism of the paper is that the doses of cocaine are very large. Braun wishes to emphasize that in the adrenalin cocaine combination *smaller* doses of cocaine can be used.

That adrenalin chloride has an inhibitory action on the general absorption of fluids cannot be doubted after the recent work of Meltzer and Auer. Klapp found that one drop of a 1 : 1000 adrenalin solution injected subcutaneously into dogs' backs would hinder the absorption of milk-sugar.

In the use of cocaine as an anæsthetic the danger is from the toxic effect due to its absorption, and it is a common observation that this does occur. The toxicity is shown by such symptoms (described by Foisy) as paleness, sweating, talkativeness, muscular tremor, delirium, and even death. It would seem, therefore, that in testing the value of adding adrenalin chloride to cocaine in its use as a local anæsthetic the effect of adrenalin on *toxic* doses of cocaine should be carefully studied. The following experiments were undertaken with this in view:

Protocols.

Experiment No. 1. Guinea-pig No. 1, weighing about 500 gm., injected with 0.5 c.c. of a 10 per cent. solution of cocaine into the abdominal cavity. After four minutes retraction of head. After eight minutes convulsions. After ten minutes death.

Experiment No. 2. Guinea-pig No. 2, weighing about 500 gm., injected with 0.5 c.c. 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later 0.06 gm. cocaine injected into the abdominal cavity. After four minutes paralysis of hind legs. After eight minutes convulsions. After fourteen minutes death.

Experiment No. 3. Guinea-pig No. 3, weighing about 500 gm., injected with 0.025 gm. cocaine into the peritoneal cavity. After thirteen minutes convulsions. Animal recovered in thirty-five minutes.

Experiment No. 4. Guinea-pig No. 4, weighing about 500 gm., injected with 0.5 c.c. 1 : 1000 adrenalin chloride solution into the peritoneal cavity. No marked symptoms. Ten minutes later

0.025 gm. cocaine injected into peritoneal cavity. Animal died in thirty-nine minutes. No symptoms of cocaine poisoning. Animal died apparently from œdema of lungs. Symptoms came on twenty minutes after injection of adrenalin.

Experiment No. 5. Guinea-pig No. 5, weighing about 500 gm., injected with 0.5 c.c. 1:1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms.

Experiment No. 6. Guinea-pig No. 6, weighing about 1000 gm., injected with 1 c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later 0.05 gm. cocaine injected into abdominal cavity. After twenty minutes well-developed paralysis of hind legs. After forty-five minutes animal had apparently recovered, but it died during the night several hours afterward.

Experiment No. 7. Guinea-pig No. 7, weighing about 1000 gm., injected with 0.05 gm. cocaine into the abdominal cavity. After eight minutes paralysis of hind legs. After ten minutes retraction of head. After seventeen minutes convulsions. Animal recovered in one hour.

Experiment No. 8. Guinea-pig No. 5, weighing about 500 gm., injected with 0.5 c.c. of a 1 : 1000 adrenalin chloride in abdominal cavity. No marked symptoms.

Experiment No. 9. Guinea-pig No. 8, weighing about 500 gm., injected with 0.5 c.c. of a 1 : 1000 adrenalin chloride into the abdominal cavity. Ten minutes later 0.025 gm. cocaine injected into abdominal cavity. No marked prostration. No convulsions. Animal recovered in thirty minutes.

Experiment No. 10. Guinea-pig No. 9, weighing about 750 gm., injected with 0.037 gm. cocaine into abdominal cavity. After ten minutes falls on side. After twelve minutes retraction of neck. No convulsions. Animal recovered in thirty minutes.

Experiment No 11. Guinea-pig No. 10, weighing about 500 gm., injected with 0.5 c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. Ten minutes later injected with 0.025 gm. of cocaine into the abdominal cavity. After fifteen minutes paralysis of hind legs. After twenty-five minutes animal seemed to partially recover but died suddenly, thirty-seven minutes after the injection of the cocaine.

Experiment No. 12. Guinea-pig No. 11, weighing about 500 gm., injected with 0.5 c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later injected with 0.025 gm. cocaine into the abdominal cavity. After fifteen minutes paralysis of hind legs. After eighteen minutes falls on side. After twenty-five minutes recovered somewhat, but died later.

Experiment No. 13. Guinea-pig No. 12, weighing about 500 gm., injected with 0.5 c.c. of a 1 : 1000 adrenalin chloride solution into

the abdominal cavity. No symptoms. Ten minutes later injected with 0.025 gm. of cocaine into the abdominal cavity. After fifteen minutes slight paralysis of hind legs. After twenty-five minutes falls on side. After twenty-seven minutes died suddenly.

Experiment No. 14. Guinea-pig No. 13, weighing about 500 gm., injected with 0.5 c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later injected with 0.025 gm. cocaine into the abdominal cavity. After fifteen minutes lies on side. After twenty-four minutes head retracted. Animal died several hours afterward.

Experiment No. 15. Guinea-pig No. 14, weighing about 500 gm., injected with 0.5 c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later injected with 0.025 gm. cocaine into the abdominal cavity. After twenty minutes paralysis of hind legs. After twenty-four minutes falls on side. Seemed to recover, but died several hours later.

Experiment No. 16. Guinea-pig No. 15, weighing about 750 gm., injected with $\frac{3}{4}$ c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later injected with 0.037 gm. cocaine into the abdominal cavity. After twenty minutes paralysis of hind legs. After thirty-five minutes falls on side. After forty minutes death.

Experiment No. 17. Guinea-pig No. 16, weighing about 500 gm., injected with $\frac{1}{4}$ c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later injected with 0.025 gm. cocaine into abdominal cavity. After fifteen minutes paralysis of hind legs. After twenty minutes falls on side. After thirty minutes retraction of neck. Animal recovered.

Experiment No. 19. Guinea-pig No. 18, weighing about 500 gm., injected with $\frac{1}{4}$ c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later injected with 0.025 gm. cocaine into the abdominal cavity. After fifteen minutes slight paralysis of hind legs. Animal seemed to recover in large measure, but died several hours after.

Experiment No. 20. Guinea-pig No. 8, weighing about 500 gm., injected with $\frac{1}{4}$ c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No symptoms. Ten minutes later injected with 0.025 gm. cocaine into the abdominal cavity. Slight paralysis of hind legs. Animal recovered.

Experiment No. 21. Guinea-pig No. 5, weighing about 500 gm., injected with 0.025 gm. cocaine into the abdominal cavity. After thirty minutes slight paralysis of hind legs. Animal recovered.

Experiment No. 22. Guinea-pig No. 3, weighing about 500 gm., injected with $\frac{1}{4}$ c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later injected with 0.025 gm. cocaine into the abdominal cavity. After

seven minutes falls on side. After twelve minutes convulsions. Animal died in twenty minutes.

Experiment No. 23. Guinea-pig No. 9, weighing about 750 gm., injected with $\frac{3}{8}$ c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later 0.037 gm. cocaine injected into the abdominal cavity. After thirty minutes slight paralysis of hind legs. Animal recovered.

Experiment No. 24. Guinea-pig No. 5, weighing about 500 gm., injected with $\frac{1}{4}$ c.c. of a 1 : 1000 adrenalin chloride solution into the abdominal cavity. No marked symptoms. Ten minutes later injected with 0.025 gm. cocaine into the abdominal cavity. After thirty minutes slight paralysis of hind legs. Animal seemed to recover, but died several hours afterward.

Experiment No. 25. Guinea-pig No. 7, weighing about 1000 gm., injected with 0.05 gm. cocaine into the abdominal cavity. No symptoms. Animal recovered.

Experiment No. 26. Guinea-pig No. 8, weighing about 500 gm., injected with 0.025 gm. cocaine into the abdominal cavity. After fifteen minutes paralysis of hind legs. After twenty minutes falls on side. Animal recovered in forty minutes.

Experiment No. 27. Guinea-pig No. 16, weighing about 500 gm., injected with 0.025 gm. cocaine into the abdominal cavity. After twenty-five minutes slight paralysis of hind legs. Animal recovered.

In order that the effects of the drugs might come on quickly and be influenced easily it was deemed best to make the injections into the peritoneal cavity of guinea-pigs. The results can be better understood by separating the experiments into groups and arranging the groups into tables. In all cases the injection was made into the peritoneal cavity. When the combination of drugs was used the adrenalin was injected first, followed ten minutes later by cocaine. The dosage was in proportion to the weight of the animal, so that each animal in the various groups received comparatively the same amount of drug. The first group consists of one experiment and its control. The dosage of adrenalin was 0.001 gm. per kilo of body weight, while of cocaine it was 0.1 gm. per kilo.

TABLE I.

Experiment No.	Injection of	Onset of symptoms in	Result.	Control.			
				Experiment No.	Injection of	Onset of symptoms in	Result.
2	Ad. + Co.	4 minutes.	Died 14 min.	1	Co.	3 minutes.	Died 10 min.

The experiment showed that the dosage of cocaine was too great, but even with this overdose the retarding action of adrenalin was manifest.

In the experiments in Table II. the dose of cocaine has been reduced to 0.05 gm. per kilo, while the dose of adrenalin is the same. Control animals in which adrenalin alone was injected were used first.

TABLE II.

Experiment No.	Injection of	Symptoms began in	Result.	Control.			
				Experiment No.	Injection of	Symptoms began in	Result.
				5	Ad.	No symptoms.	Recovered
				8	Ad.	"	"
4	Ad. + Co.	10 min.	Died	3	Co.	8 min.	"
6	Ad. + Co.	20 "	"	7	Co.	8 "	"
9	Ad. + Co.	No marked symptoms.	Recovered	10	Co.	10 "	"
11	Ad. + Co.	15 min.	Died	17	Co.	No symptoms.	"
13	Ad. + Co.	15 "	"				
14	Ad. + Co.	15 "	"				
15	Ad. + Co.	20 "	"				
16	Ad. + Co.	20 "	"				

The above table shows a remarkable record. Not an animal of the series which received the dose of cocaine alone died, while only one of the adrenalin-cocaine animals lived. By referring to the protocols it will be found that animals Nos. 6, 12, 14, and 15 seemed to recover in large measure, but died later. Animal No. 4 died apparently of œdema of the lungs, but it was the only animal in the whole series of experiments that showed any marked effect from the dosage of adrenalin. It will be noticed that the symptoms come on somewhat quicker in the animals that were injected with cocaine only.

In Table III. the experiments were the same as above except that the dosage of adrenalin has been reduced to 0.0005 gm. per kilo. The reduction in the amount of adrenalin was made on the supposition that, inasmuch as the animals were receiving almost a lethal dose of both adrenalin and cocaine, the combination proved fatal.

TABLE III.

Experiment No.	Injection of	Symptoms began in	Result.	Control.			
				Experiment No.	Injection of	Symptoms began in	Result.
18	Ad. + Co.	15 min.	Recovered	25	Co.	No symptoms.	Recovered
19	Ad. + Co.	15 "	Died	26	Co.	20 min.	"
20	Ad. + Co.	25 "	Recovered	27	Co.	25 "	"
21	Ad. + Co.	30 "	"				
22	Ad. + Co.	7 "	Died				
23	Ad. + Co.	30 "	Recovered				
24	Ad. + Co.	30 "	"				

Before making any conclusions from these experiments it should be noted that all of the animals except Nos. 18, 19, and 21 had had previous injections of cocaine. The possibility of a slight immunity having been established should be considered. Three out of the seven adrenalin cocaine animals died while none of the control animals died.

What then are the conclusions to be drawn? The same criticism holds here that Braun made of Thies' work. The experiments are made with too large doses of cocaine to show the real value of adrenalin cocaine as a local anæsthetic. What is certainly shown is that *adrenalin will not protect the organism against toxic doses of cocaine.*

In the use of adrenalin cocaine care should be exercised not to inject a toxic dose of the latter, for not only does the adrenalin fail to protect the body against the toxic doses of cocaine, but it seems to enhance the toxic action.

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THE INFLUENZA BACILLUS IN BRONCHIECTASIS.

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That dilatation of the bronchi may occur in the course of acute influenza, either of the bronchial or pneumonic type, has been demonstrated by necropsy findings and the cultivation of the specific organism from the lesions. (Leichenstern, M. Beck, and others.) Similar cases have also been reported in which recovery from the acute illness occurred with the gradual development of chronic bronchitis or bronchiectasis.

In our own country the relationship of bacillus influenzæ to chronic pulmonary disease has received but little attention, if we except the extensive investigations of Lord.

It may therefore be of interest to present the findings in some cases observed at the Johns Hopkins Hospital (service of Prof. Osler) during the early part of 1905.

CASE I. *Total duration three months; onset with symptoms of acute coryza; gradual development of cough and profuse expectoration; small hemorrhages, irregular temperature, sweats; influenza bacilli in pure culture from sputum; headache, partial paralysis; death; autopsy; diffuse bronchiectasis, bronchial erosion, patches of bronchopneumonia, small foci of necrosis; influenza bacilli in pure culture from tissues; no tuberculosis.*—B. R. F., male, white, aged twenty-nine years, trained nurse. Admitted February 16, 1905, complaining of "cough and pains in chest."

Family History. Good; negative for tuberculosis.

Past History. No acute diseases of childhood; never any pneumonia; no typhoid fever; no hemoptysis; severe la grippe three years ago; lasted two months; no other pulmonary trouble until present illness; no history of gastric or intestinal disease; no renal troubles; primary syphilitic sore five years ago; no secondaries; always strong and healthy; moderate use of alcohol; heavy smoker of cigarettes; average weight 145 pounds.

Present Illness. Two months before admission patient caught cold after exposure. This was at first slight, not accompanied by cough. It slowly grew worse, however, and there has been severe cough with increasingly profuse expectoration; for about three weeks patient has felt feverish at night. In past four days the cough much worse, with nocturnal dyspnœa, fever, sweats, and pain in right side. He has lost fifteen pounds in weight since onset.

Physical Examination. On admission patient was well nourished and not in great distress. Diminished expansion more marked on right side. Vocal fremitus impaired, with dulness on percussion over right axilla and lower back and over left lower back. Breath sounds enfeebled over dull areas, though nowhere absent. No tubular breathing. No friction rub. No movable dulness. Very numerous moist and sonorous rales over entire chest. Heart sounds clear and of normal relative intensity. Abdomen normal; deep reflexes normal. Temperature irregular, varying in first twenty-four hours from normal to 100.5°. Pulse soft, regular, 96 to minute. No marked thickening of vessel walls. Sputum profuse, greenish, mucopurulent, with a sickening, sweetish odor. Examination for elastic tissue and tubercle bacilli negative. Exploration of chest with needle yielded no results. Leukocytes on admission, 22,000; hæmoglobin, 90 per cent. Patient did not improve, paroxysms of coughing grew worse.

February 24th. First culture from sputum absolutely pure growth of bacillus influenzæ.

25th. Small hemorrhage from lungs, followed by chill and sweat. Pulmonary signs remained about as at first, except for increase in sonorous rales. Leukocytes varied between 12,000 and 25,000.

March 8th. Patient complained of numbness and some apparent weakness in right hand. Some headache. Two days later pain in

right testicle, with increased weakness of wrist and muscles of leg on same side; no absolute paralysis, but marked wrist-drop and great weakness of whole right side. No changes in sensation. Testicle objectively normal. Severe and constant headache. No irregularity of pupils. No stiffness of neck. No Kernig's sign. The patient gradually became weaker. On March 18th became very cyanotic and seemed unable to clear the lungs of the accumulated sputum. Signs in the chest were unchanged, except for the increase of loud bubbling rales.

19*th*. Blood cultures on various media entirely sterile. Patient died March 20th. Clinical diagnosis: bronchiectasis.

Autopsy. Both pleural cavities partly obliterated by old adhesions, mostly posterior. Lungs voluminous; surface roughened by fresh fibrinous exudate. On section the lung is foul smelling and studded throughout with irregular patches, which are prominent and rough on cut surface. These patches have usually a yellowish-white centre, surrounded by grayish-red areas. At one point in upper right lobe there is a small cavity with a necrotic lining and surrounded by an area of consolidation.

The bronchi are dilated and contain a considerable quantity of a thick, semi-fluid material. The trachea is much inflamed and the main bronchi have a circumference of 4 cm. below the first division. The bronchi in the greater part of the lung are not very greatly dilated, but have thickened walls. Toward the periphery of lungs, however, the smaller bronchi are dilated into spindle-shaped enlargements like beads on a necklace. These bronchi are dark red and apparently eroded. The bronchi to the upper lobes are less involved and cleaner than those below.

Microscopic examination shows numerous areas of bronchopneumonia tending to undergo necrosis in the centre. Necrosis frequently involves alveolar walls and adjacent bronchus. Considerable masses of bacteria and leukocytes may be seen in the central portions of these foci. Many bronchi are entirely eroded in places, the erosion extending to the alveoli, so that quite large cavities are formed, no trace of the bronchial wall remaining. In other parts the bronchi are only partly eroded and are very thin and dilated. The larger bronchi are not so extensively destroyed, showing merely desquamation of epithelium and infiltration of cells. The enormously enlarged bronchial lymph glands are œdematous, their sinuses filled with fluid, but not containing an excess of cells. The lymph cords are increased in bulk and the bloodvessels dilated. No areas of necrosis are present.

Permission for examination of the brain and spinal cord refused.

Cultures from contents of bronchi yielded bacillus influenzæ in great number with a few streptococci. From the washed and teased bronchial wall pure cultures of bacillus influenzæ were obtained, as also from the gangrenous areas. The lymph glands were sterile.

The bacterial masses above noted were seen in sections stained with methylene blue and eosin or with carbol thionin to consist almost entirely of tiny rods resembling the influenza bacillus. The infiltrating cells about the bronchial epithelium and extending down into the wall were frequently packed with these tiny rods. A few cocci and large bacilli were seen on the surface, but not deep in the tissues.

This case shows many of the features ascribed to acute influenza in the writings of Pfeiffer, Leichtenstern, Hitzig and others. The duration and slow development in this case would seem, however, to place it with the subacute or chronic cases. Here we have every reason to believe from cultural evidence during life and at postmortem that bacillus influenzæ was the principal if not the exclusive cause of the extensive pulmonary changes.

The case was in many ways clinically like a rapidly progressing phthisis, with the irregular fever and sweats, the loss of weight, and profuse expectoration and hemorrhage. Yet the daily examination of the sputum revealed no tubercle bacilli; and at autopsy the most careful search for tuberculous lesions, macroscopically and microscopically, was negative. The nervous features of the case strongly suggest the hemorrhagic encephalitis or "encephalitis grippalis" described by Leichtenstern, Königsdorf, Fürbringer, and others in association with influenzæ. In the absence of postmortem observations on the brain, however, the diagnosis can but be provisional. Prof. Osler suggested that brain abscess might account for these features of the case.

CASE II.—*Cough three years; worse in last year; large amount of foul sputum coughed up in paroxysms; emphysema; orthopnœa; no fever, chills, or sweats; no hemorrhages; bacillus influenzæ cultivated from sputum in practically pure culture; progressive weakening; steady increase in amount of expectoration; death; autopsy.*—B. G., male, white, aged forty-seven years, bookkeeper. Complains of "bronchitis." Admitted December 14, 1904. Died February 4, 1905.

Family History. Unimportant. No pulmonary disease.

Past History. Had scurvy in Africa with enlargement of liver; no pneumonia; no hæmoptysis; no night sweats; "severe catarrh" in 1893 and again in 1895; no other history suggesting influenza; habits good.

Present Illness. Began to cough three years ago. Worse last two years. Foul odor increasing and large quantities of sputum in paroxysms of coughing for a year past. Very severe paroxysms on rising, with evacuation of large amounts of sputum, greenish, purulent, rather thin and frothy. Paroxysms occurring several times a day and interfering with sleep at night. No hæmoptysis; no chills or sweats; no pain; has lost steadily in weight and strength in past year.

Physical Examination. Skin slightly cyanotic. Some clubbing of fingers. Chest rounded. Marked dyspnœa with activity of

accessory muscles of respiration. Percussion note hyperresonant throughout chest, except in back, where there is slight impairment over left base and right interscapular region. Breath sounds everywhere well heard, and accompanied by numerous moist and piping rales. No tubular or amphoric breathing. No cavity made out. No friction rub. Blood count showed 4,600,000; erythrocytes, 9600; leukocytes and hæmoglobin of 87 per cent.

Bacillus influenzæ in very large numbers were cultivated from the sputum, with a very few colonies of diplococcus pneumoniæ. The patient did not respond to treatment and favorable surroundings. His cough continued severe and the amount of sputum large. There was never any elastic tissue found and daily examinations for tubercle bacilli were negative. The patient gradually lost strength, and was more and more exhausted by the coughing paroxysms necessitated by the profuse expectoration. He died February 24, 1905, without any acute exacerbation of his trouble.

Autopsy. Both lungs bound by old adhesions posteriorly. Bronchi contain a frothy turbid fluid with sickening odor. Lung substance emphysematous. No areas of consolidation. Bronchi everywhere dark red, granular-looking, and extremely and almost uniformly dilated to periphery of lungs. No special saccular dilatations or cavities. No tubercles.

Microscopically, there was general emphysema. Epithelial lining of bronchi well preserved in the main and showing scattered areas of metaplasia into a stratified squamous type with many mitoses. Marked infiltration of mucosa with lymphoid cells. The epithelial layer invaded by polymorphonuclear leukocytes. Hyperplasia of peribronchial lymph follicles. Atrophy of muscular coat and increase of the peribronchial connective tissue, including in places atelectatic alveoli with cubical epithelium. No evidence of tuberculosis. Owing to the fact that the lungs were injected with formalin *in situ*, it was impossible to secure cultures from the tissues at autopsy.

Stained sections, as in the previous case, however, showed that the walls of the bronchi were infiltrated with leukocytes, containing great numbers of small bacilli like bacillus influenzæ. Very few pneumococci were found in the sections stained by Gram's method. Coarse non-Gram staining rods were found in the masses of secretion in the bronchial lumen. The intracellular organisms were all of the influenza type.

CASE III.—*Chronic cough for thirty years; acute influenza eleven years ago with increased paroxysmal cough and profuse expectoration; signs of cavity in both lungs; no hæmoptysis; no tubercle bacilli; no elastic tissue; influenza bacilli in pure culture from sputum; clubbed fingers; arthritis deformans.*—S. N., single, white, female, aged forty-nine years, seamstress. First admitted in June, 1899. Complained of cough and profuse expectoration.

Family History. Good; no tuberculosis.

Past History. Subject to colds and winter coughs from girlhood. Measles the only acute disease of childhood. "Rheumatism" for seven years past, with gradual stiffening of fingers, shoulders, knees, and hips. In 1894 patient had severe attack of la grippe, since which time cough has been worse and expectoration more profuse. No night sweats; no hæmoptysis.

Present Illness. Really dates years back, a history of chronic paroxysmal cough with exacerbations every winter. Sputum abundant, tenacious, greenish, foul.

Physical Examination. A thin, poorly-nourished woman. Well-marked arthritis deformans, involving both knees and elbows; slight involvement of shoulder-joints.

Lungs. Signs of diffuse bronchitis over both fronts. Distinct signs of cavity in both interscapular regions; signs varying before and after paroxysms of cough and expectoration. Numerous moist and sonorous rales throughout both backs and axillæ. No fever; no sweats. Daily examination of sputum negative for tubercle bacilli and elastic tissue.

Second Admission, January 18, 1905. Complaint "cough." Arthritis more marked, involving nearly all the joints to a greater or less extent. Well-marked spondylitis. Cavity signs (amphoric breathing, pectoriloquy, cracked-pot sound, etc.), definite in both interscapular regions where previously noted. Diffuse moist sonorous rales. Large amount of fetid sputum. No hæmoptysis. Bacillus influenzæ in enormous numbers cultivated from sputum. Also small numbers of pneumococci. Sputum constantly negative for tubercle bacilli and elastic tissue.

Cultures from joint fluids negative on all media.

CASE IV. *Twelve years' standing; acute onset; probable empyema; early cavity formation; thickened pleura; paroxysmal cough; profuse foul expectoration; severe recurrent hemorrhages for ten years past; marked scoliosis; extreme hypertrophic osteoarthropathy; exacerbations often attended by fever and sweats; no tubercle bacilli; no elastic tissue; influenza bacilli in sputum in great number and pure culture.* —H. H., white, male, aged twenty-nine years, baker.

Family History. Unimportant; no tuberculosis.

Past History. Always strong and well until onset of present illness. Never any pulmonary disease. Habits good. Hands and feet unusually large from babyhood. In December, 1893, patient had severe cold after exposure; pain in right side; chills and fever; cough; night sweats. Profuse greenish expectoration; never blood-tinged. In bed two months. After patient was up and about for two weeks he began to have severe paroxysms of cough in the morning, bringing up large amounts of yellowish-green sputum, rather sweetish and sickening, probably due to the discharge of an empyema through the lung. This paroxysmal cough persisted, and fifteen months later the patient had his first hemor-

rhage from the lungs and was admitted to the hospital in March, 1895.

On this admission the patient was emaciated and very pale. Great enlargement of bones of hands, feet, and forearms. Right shoulder lower than left. Right chest rather flat above. Left lung clear, except for scattered rales. Right side shows definite signs of much thickened pleura below fifth rib and suggestion of cavity at angle of scapula. Sputum abundant, bloody. No tubercle bacilli or elastic tissue.

In the ten years following the patient was readmitted sixteen times, on nearly every occasion for severe hemorrhage from the lungs. The deformity of chest and right lateral scoliosis became more marked. Signs of cavity quite definite in right scapular region. Densely thickened pleura on right side. Diffuse rales on left. Osteoarthropathy more marked. Patient had persistent paroxysmal cough and profuse expectoration, rather fluid and purulent, very foul at times. Fever and sweats in the periods of exacerbation. Between these more severe attacks patient able to work at his trade. Patient responded well to treatment (rest, good food, etc.), cough becoming much less frequent and sputum greatly diminished in amount. There was always at least one paroxysm in a day, however.

Last admission, February, 1905. Condition practically unchanged except for a dry pleurisy on left side. Influenza bacilli were obtained in absolutely pure culture from sputum and in enormous numbers at first. As patient's condition improved the number of bacteria greatly decreased, though there were always a few to be found. In the ten years' observation there was never any elastic tissue found nor any tubercle bacilli.

Intratracheal injections were particularly beneficial to this patient, causing rapid diminution in amount of sputum and great reduction in number of bacteria, a 10 per cent. suspension of iodoform in olive oil being used for this purpose.

CASE V.—*Cough for nine years; history of pneumonia; empyema; operation; contraction of pleura; gradual development of paroxysmal cough with profuse, very foul expectoration; right lateral scoliosis; exacerbations with fever and sweats; no hæmoptysis; no tubercle bacilli; pneumococcus from sputum with small proportion of influenza bacilli and a fetor-producing organism; operation for drainage of cavity.*—R. S., white, female, aged fifteen years, school-girl.

Family History. Unimportant.

Past History. No illness other than chickenpox till an attack of pneumonia (right side) at age of six years, followed after twelve weeks by empyema and operation. Since this time patient has had a chronic paroxysmal cough with profuse, very foul expectoration, worse in winter, often prostrating her. Never any hæmoptysis. On first admission, October, 1899, patient was found to have definite

signs of cavity in posterior and upper part of right lower lobe, there being a very noticeable intensification of the signs after the paroxysms of coughing. Left side clear, except for a few coarse rales. Some clubbing of fingers. Well-marked scoliosis to right. Sputum very profuse, very foul, of a greenish color. No tubercle bacilli or elastic tissue found. In the succeeding six years the patient was frequently readmitted with exacerbations of cough and expectoration, usually with some fever and occasional sweats; cavity signs in right lung persisting. She developed quite well, despite her infirmity. The cough and fetor were very distressing, however.

Last admission, January 19, 1905. Physical signs unchanged. Cough very distressing. Sputum from 200 to 400 c.c. in twenty-four hours. Influenza bacilli constantly present in sputum, but in small numbers, both on coverslips and in cultures. Pneumococcus in much greater preponderance. There was also isolated on one occasion a small micrococcus, which decolorized by Gram's method and grew only on blood-containing solid media, as will be noted later.

Patient was transferred to the surgical side and operation for drainage of the cavity in right lung was performed on May 23, 1905, by Dr. J. C. Bloodgood. Several ribs were resected and the lung exposed over the area of cavernous breathing. The lung was very atelectatic and dark red-like liver. Several small cavities were incised and drained. Patient is doing well two weeks after operation. Cultures from the contents of the cavities at operation yielded pneumococcus in great preponderance, with a few colonies of bacillus influenzæ. The surgical and pathological aspects of the case will be more fully reported by Dr. Bloodgood.

In addition to the above five definite cases of bronchiectasis there were two others of chronic bronchitis with probable dilatation of bronchi, from both of which influenza bacilli were obtained in absolutely pure culture. Both were men in middle life; had history of cough and profuse expectoration for years, worse in winter. Often fever and rarely night sweats. No hæmoptysis. In neither case could any tubercle bacilli be demonstrated by daily examinations.

A sixth case which has a bearing on the pathogenicity of bacillus influenzæ as found in the sputum in chronic cases will complete the list.

CASE VI. *Chronic bronchitis thirty years; slight hæmoptysis; localized pleurisy with encapsulated effusion; bacillus influenzæ in pure culture from sputum; two weeks later empyema with bacillus influenzæ in pure culture from pus; operation; recovery.*—A. B., male, white, aged sixty years, tailor.

Family History. Negative.

Past History. Always healthy, except for chronic cough, worse in winter. No history of any acute pulmonary disease preceding.

Present Illness. In the winter of 1904 the patient had a severe attack of bronchitis; spat a little blood; occasional fever and sweats.

Rather profuse expectoration. Never quite well since. Four weeks ago became much worse, pain in side, night sweats, hæmoptysis slight.

Physical Examination. Diffuse bronchitis. Slight movable dullness in lower left axilla and back; irregular fever and sweats. Small amount of fluid, straw-colored, obtained by aspiration. Cultures on ordinary media sterile. Septum abundant, tenacious, mucopurulent blood-streaked. Cultures from sputum on March 6th yielded bacillus influenzæ in absolutely pure culture. Patient discharged on request March 12th, not much better. Readmitted March 30th in practically same condition, though feebler and with more fever and drenching sweats. Moderate leukocytosis. Signs of pleural effusion. Small amounts of slightly bloody fluid aspirated on several occasions. This was, unfortunately, not saved for cultures. On April 11th a few cubic centimetres of pus obtained by aspiration and from this influenza bacilli grew in absolutely pure culture. Patient transferred to surgical side. Eighth rib resected and pleura found very thick. A considerable amount of grayish, odorless pus evacuated and cavity drained. Patient improved rapidly.

The bacillus of Pfeiffer seems to have been the direct cause of the empyema in this case, the sputum having yielded absolutely pure cultures of this organism for some time previous to the onset of the empyema, and the same organism, and it alone, being found in the pus from the pleura at operation by culture and cover-slips.

Cultural Methods. The sputum was collected in sterile cups and examined without delay. The washing method employed was substantially that described by Kitasato and by Lord. It was interesting to observe the striking difference in the bacteria found on coverslips made before and after washing. These were stained by Gram with bismarck brown counterstain in exactly the same way. While those made before washing showed usually a greater or less number of extracellular organisms (diplococci, streptococci, and other mouth bacteria), in addition to bacillus influenzæ, it was quite possible in most cases to wash the sputum so that the particles now showed only influenza bacilli, and these all intracellular. Pigeon-blood agar was used in most cases as a culture medium; in others sterile fetal blood from the placenta was added to the agar. On this latter medium it was interesting to note that involution forms appeared earlier and in much greater number than when pigeon, rabbit, or adult human blood was used. Some of these forms were so large and irregular as to give at first the impression of a mixed infection. Transfers to fresh media and control cultures of the same size and source on other blood media, however, produced in every case a pure typical growth of bacillus influenzæ. The gross appearance of the colonies on the fetal-blood agar was not different from that on the other tubes, being always the tiny, water-clear,

discrete colonies, exactly as described by Pfeiffer. The small Gram decolorizing micrococcus isolated from Case VI. has not been identified accurately. It grew only on solid media containing blood. It decreased rapidly in vitality, so that it was lost after the fifth or sixth transfer. Its most striking characteristic was the production on the blood plates of the intense sickening sweetish fetor common in bronchiectasis. In some respects it conforms to micrococcus catarrhalis though that organism grows on ordinary media, and I have been unable to find any reference to its production of fetor when grown upon blood agar. None of the other organisms grown from the sputum in these cases (bacillus influenzæ, streptococcus, or pneumococcus) produced any fetor in pure or mixed cultures on like media.

CONCLUSIONS. 1. The influenza bacillus is probably capable of producing extensive pathological changes in the lungs, leading at times to bronchiectasis (Cases I. and IV.). The organism may be a secondary invader or associated with other bacteria; the latter may be the preponderant factor. In our small series there was no clinical difference determinable between the pure influenza infections and the mixed or non-influenza cases.

2. The very close clinical resemblance of these cases of bronchiectasis to some cases of chronic tuberculosis with cavity formation is important, and may, in the absence of cultural investigations, lead to wrong diagnoses. Physical signs alone, therefore, cannot be relied upon for a positive diagnosis of tuberculosis.

3. Care should be taken to prevent the possible infection of others from these chronic cases having influenza bacilli in the sputum, as the organisms may not have lost their pathogenicity by a continued growth in these old infections.

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N. B.—Since the completion of this investigation, and while waiting for surgical interference in Case V., a second admirable and comprehensive paper has appeared by Dr. Lord (Boston Medical and Surgical Journal, May 11 and 18, 1905), which covers a much wider field and greater experience. With his conclusions my smaller series seem fully to agree.

REVIEWS.

INTERNATIONAL CLINICS. Edited by A. O. J. KELLY, A.M., M.D., Philadelphia, Pa. Vol. I., fifteenth series, pp. 312. Philadelphia and London: J. B. Lippincott Co., 1905.

THIS quarterly, containing many papers of interest to the advanced practitioner, comes as a welcome visitor. Opening with a practical presentation of cardiac asthma by Merklen, followed by a medical clinic by Robin, and an article on infected cholelithiasis by Chantford, we obtain a fair view of the modern French practice. Douglas offers a scientific study of the carbohydrates in Human Urine in Health and Disease, while the eye and hand in the Diagnosis of Heart Disease by Walsh, who is always suggestive, and Parkinson on the Early Diagnosis of Heart Disease in Childhood, and Manges on Aortic Stenosis and Adherent Pericardium would satisfy the internalist even if Kendict, in an excellent discussion on Intestinal Adhesions, were not added for good measure. In surgery Young presents a description of a valuable modification of Wolfe's or Krante's method in skin grafting. Dawbarn, in a characteristic, forcible, and practical article, writes on the Starvation of Malignant Growths by Depriving them of Blood Supply. Durante's new Operative Method for the Total Extirpation of the Larynx seems formidable, but if it obviates the danger of inhalation pneumonia, should be made use of. Hibbs formulates the treatment of knee-joint disease from the wealth of material at his disposal. Glenard's disease is likely to come more prominently, if it be needed, into the field of the physician through the very sensible article by Gallant.

The thoughtful general practitioner will find much that he recognizes in his daily work in the paper by Mills on Morphinomania, Cocamania, and General Narcomania and some of their legal consequences, for it is to him that such patients come at a time when unfortunate consequences are more easily prevented. Carr adds to the increasing list of instances of cerebellar tumor, and Bramwell gives an excellent presentation of two cases of ocular palsy probably caused by a lesion near the thermoidal fissure. The single paper on Anterior and Posterior Parietal Presentations of the Head in Slightly Flattened Pelves, by Jardine, completes the list of individual articles. The Progress of Medicine for 1904 occupies more than a hundred pages. Stevens presents Treatment, Edsall and Stanton

Medicine, and Bloodgood Surgery. We are pleased to see that treatment is not to be taken as synonymous with medicine, but that it covers a wider field than medicine and surgery combined. And to one who may practice in a narrower field than these two great divisions of the healing art we commend this section for the wisdom of the choice of a reviewer whose broad reading is reflected by the entire section. From this far too brief summary of the contents of this volume it is evident that the usefulness of this series is fully maintained and in source the papers are actually international.

R. W. W.

ORGANES GENITO-URINAIRES DE L'HOMME. Par HENRI HARTMAN. 8vo., in five parts, with 412 illustrations. Paris: G. Steinheil, editeur, 1904.

THIS volume thoroughly covers the subject and is up to date in the newer phases thereof, notably in the newer methods of urethroscopy and cystoscopy. The older parts of the subject are thoroughly gone into, notably the preparation and care of instruments, exploration of the urethra, prostate, and bladder. The chapter on injections, lavage, and instillation, while presenting nothing new, is in accord with our own authors, but the illustrations on urethroscopy and cystoscopy are profuse and easily understood. Part II. deals with the treatment of acute and chronic urethritis, foreign bodies in the urethra, and operations thereon, thoroughly and concisely. The operations on the urethra are considered from the point of operation, as perineal, rectal, and hypogastric, and here cystotomy and cystostomy are also considered. Lithotrity and Bottini's operation each have a separate chapter and are admirably treated. The treatment of ailments of the urethra, bladder, and prostate follow in the main the principles laid down therefor in this country. The therapeutics of ailments of the kidney, ureter, and pelvis contain minute direction for the physical examination of the kidney as also its functional examination, although the method advised for separate ureterization seems crude in the extreme and not equal to our own. Puncture, nephrectomy, nephrotomy, nephrolithiasis, nephrostomy, nephropexy, their treatment, operation, and accidents due to them, are thoroughly considered, as is the surgery of the ureter and pelvis. Part IV. considers the congenital organs, traumatisms, castration, epididymectomy, anastomiasis of the spermatic cord, here quoting Martin's case operated on at the University Hospital of this city. The treatment of hydrocele, varicocele, hæmatocele, and cysts of the cord and epididymis, as well as vaginitis, orchitis and elephantiasis, tuberculosis and syphilis of the testicle, follows the usual lines. The penile and pre-

putial conditions, the operation, together with the consideration of the vices of formation, occupy the rest of the volume, and aside from the illustrations, which are above the average, present nothing new and follow the usual lines. E. A. H.

DISEASES OF THE SKIN: THEIR DESCRIPTION, PATHOLOGY, DIAGNOSIS, AND TREATMENT, WITH SPECIAL REFERENCE TO THE SKIN ERUPTIONS OF CHILDREN, AND AN ANALYSIS OF FIFTEEN THOUSAND CASES OF SKIN DISEASE. By H. RADCLIFFE-CROCKER, M.D. (Lond.), F.R.C.P., Physician for Diseases of the Skin in University College Hospital; Honorary Member of the American Dermatological Society; Membre Correspondant Etranger de la Société Française de Dermatologie, etc. Third edition, revised and enlarged, with 4 plates and 112 illustrations. Philadelphia: P. Blakiston's Son & Co.

THIS new edition of Crocker's *Diseases of the Skin* is a ponderous volume of more than 1400 pages, half again as large as its predecessor. A long list of subjects not included in previous editions is considered, of which the most important are *x*-ray dermatitis, toxin serum eruptions, porokeratosis, granuloma pyogenicum, leukæmia, and pseudoleukæmia cutis, acne necrotisans, acne agminata, folliclis, folliculitis decalvans, and blastomycosis hominis. We note with hearty approval that the author, for the sake of uniformity of nomenclature, has changed the names of some of the diseases; and we sincerely hope other authors, influenced by this example, may likewise endeavor to bring some order out of the chaos of names in which dermatology has so long floundered.

Concerning the role of parasites in the causation of eczema the author's view is that "while a limited number of cases of local dermatitis indistinguishable from eczemas are parasitic, in most the dermatitis, however caused, only opens the door to the parasites whose presence keeps up local irritation, so that their destruction is an important step in the restoration of the skin *ad integrum*." He believes that toxins, having their origin most frequently in the alimentary canal, often cause eczema, although he admits the difficulty of definitely proving it. He accepts unreservedly the view that seborrhœic dermatitis, Unna's seborrhœic eczema, has a parasitic origin.

Psoriasis is believed to be due to an unknown microparasite which finds conditions suitable for its growth in the skins of certain individuals only. This parasite, while reaching the skin from without, probably reaches the circulation in a certain number of cases, causing the wide and rapid spread of the eruption which is not infrequently observed. In the treatment of this annoying and obsti-

nate affection the author accords a prominent place to salicin and its derivatives, remedies which he introduced to the notice of the profession in 1895. Salicin is preferable to the salicylates because it is much less apt to disturb the stomach. In doses of 15 to 20 grains three times a day it usually produces paling of the patches and a noticeable diminution of the scaling; and, unlike arsenic, it may be given in the acute stages of the disease without danger of increasing or aggravating the eruption.

Some 55 pages are devoted to the discussion of tuberculosis of the skin. This great increase in the space given to this most important subject, while due in large part to the numerous additions made to our knowledge in the past decade, is not wholly so, but results in part from the rearrangement of the subject-matter of the text by which certain diseases are now included among the tuberculous affections which formerly were given a place apart, as, for example, the scrofulodermata. The author seems only mildly enthusiastic concerning phototherapy in lupus vulgaris, and apparently is not yet quite ready to throw aside all the older methods of treating this distressing malady for the new one; he even believes there is still a place, although a limited and subordinate one, for the greatly discredited tuberculin.

Perhaps the most extensive alterations of and additions to the text are to be found in the sections on the vegetable parasitic diseases. These embody the very extensive increase in our knowledge of the ringworm fungi resulting from the work of Sabouraud and others during the ten years which have elapsed since the appearance of the second edition.

A short section is devoted to blastomycosis, an interesting and important parasitic affection of the skin whose clinical and pathological features have been made known to the profession chiefly through the work of American observers.

In conclusion, the same praiseworthy features which early won for this treatise an enviable place in dermatological literature, characterize this new edition, and we heartily recommend it to all those (and this should include every member of the profession) who are interested in this department of medicine. M. B. H.

MALADIES MICROBIENNES EN GENERAL. Par PAUL CARNOT. Nouveau Traité de Médecine et de Thérapeutique. Publié en fascicules, sus la direction de MM. P. BROUARDEL et A. GILBERT. Paris: T. B. Baillière et Fils, 1905.

THIS volume of two hundred and odd pages represents the first "fascicule" of the "Nouveau Traité de Médecine," edited by Brouardel and Gilbert. The book is to be published in pamphlet form.

Each pamphlet is to deal with one subject, and it is expected that the forty sections which go to form the system will appear in rapid succession until the work is completed. The present "fascicule" is evidently intended as an introduction or preface to the later monographs, for the subject of bacterial diseases is treated in the broadest possible manner and does not admit very well of detailed review. The work is not even divided into chapters. The subject receives very free treatment and forms itself into an expanded introduction to bacteriology, pathology, medicine, and therapeutics. The book is roughly divided into several parts: the comparative anatomy and physiology of the microbe and organic cell; the general etiology of infections; the general pathology of infections, including paragraphs on the virulence of bacteria, and the modern conceptions of immunity. The general pathological anatomy of infections, containing paragraphs on cellular degenerations and regenerations the general symptomatology of infections; the general diagnosis and prognosis of infections, and the general therapeusis of infections. None of the subjects are developed in detail, and even the question of immunity receives but short treatment. The results of modern investigations are very freely quoted and the authors' names mentioned, but there is unfortunately no bibliography. W. T. L.

LEHRBUCH DER KINDERKRANKHEITEN FUR AERZTE UND STUDIRENDE, VON DR. ADOLF BAGINSKY, a. o. Professor der Kinderheilkunde an der Universität Berlin, Director des Kaiser und Kaiserin-Friedrich-Kinderkrankenhauses. Achte völlig neu durchgearbeitele und verbesserte Auflage. Leipzig: Verlag von S. Hirzel, 1905.

THE demand for an eighth edition of Baginsky's *Lehrbuch* is a well-deserved compliment to the author and serves to emphasize the long-acknowledged excellence of this widely known classic. In the present edition the text has been thoroughly revised and some minor changes have been made in the systematic grouping of the sections on the diseases of the newborn, the gastrointestinal diseases, and the diseases of the special sense organs. Practically there was little to change in the already well-revised text of the preceding edition. As it now stands it must be acknowledged to be the most systematic, complete, and encyclopædic treatise on the diseases of children in any language, and as such it constitutes an indispensable adjunct to the library of every physician whose practice includes the care and treatment of children. T. S. W.

PROGRESS

OF

MEDICAL SCIENCE.

MEDICINE.

UNDER THE CHARGE OF

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1. **Double Heart Rate, with Remarks on the Analysis of Irregular Pulses.**—HOFFMANN (*Zeitschrift f. klin Med.*, 1904, Bd. liii. p. 206). 2. **The Paroxysmal Occurrence of Doubled Heart Rate.**—LOMMEL (*Deutsch. Arch. f. klin. Med.*, 1905, Bd. lxxxii. p. 495). 3. **Contribution to the Study of the Extrasystoles.**—GERHARDT (*Ibid.*, p. 509).—These three contributions present the most recent views on paroxysmal tachycardia. Hoffmann was able to obtain numerous pulse tracings at the beginning and termination of attacks, and makes the following interesting observation: With a pulse of a normal period of $\frac{4.6}{5}$ second, suddenly occurs a diminution to $\frac{2.3}{5}$ second, with each second beat much smaller in size than the preceding; after about seven such beats a still smaller one begins to be interpolated just in the middle of each beat, and this rapidly growing in size, the waves soon recur at regular intervals of $\frac{1.15}{5}$ second. These waves, it is true, are not all of one height, but the variations have no regular recurrence and the interpolated waves become indistinguishable from the original $\frac{4.6}{5}$ elevations. In other words, a pulse of 60 per minute rises suddenly to 120 and then rapidly to 240, so that the first rise is just twice and the second four times as rapid as the original normal pulse. The termination of an attack does not, as a rule, occur with such precision; the pulse becomes irregular and long and short periods vary for about three beats until the rate drops to normal. Even this irregularity, however, is remarkable, as the length of each individual beat represents a multiple of the length of the foregoing rapid beats. The venous pulse, too, during this decline is of great interest, as it shows distinctly that the auricle beats at the increased rate longer than the ventricle. Hoffmann goes over the literature carefully and selects many cases which he is sure must, if carefully observed, have shown the same condition as his own.

Such a proportionate increase and decrease in the rate of contraction of the cardiac muscle is not without analogy in physiological experiments. Kries has shown that by warming and cooling different parts of the heart the rate of contraction of the warmed part is always a mul-

tiple of 2 of the rate of the cooled part. Thus it may be 2, 4, 8, 16 times as rapid, but never 3, 5, or 7, etc. Gradual changes in the temperature were always associated with sudden changes in the rate, N beats going at once to N 2 beats and then to N 4 beats, etc. Engelman, also, has noticed in pieces of resected vein from the frog's heart that sudden single stimuli thrown in at the end of diastole will cause the muscle to make a number (from 2 or 3 to over 50) of rapidly succeeding contractions, the new rate being just double the original, while the return to the original rate is abrupt, not gradual, as in stimulation of the accelerator nerves. The origin of the cardiac impulse has been definitely placed in the vena cava, and it may be assumed that there is formed here a continuous and constant stimulation always ready to act but able to manifest itself only periodically on account of the refractory period of the muscular contraction, or that the stimulating substance is itself used up in discharging an impulse and must reaccumulate before being able to start a new contraction. As evidence favors the second view, changes in contraction rate should then depend upon positive or negative chronotropic[1] influences. The theory of a constant stimulus would involve the assumption of the existence of negative or positive changes in irritability. Either of these views explains satisfactorily enough the usual variations in heart rate, but there would appear to be no reason why they should act so as to cause abruptly a halving or doubling of the previous frequency. Analogy with physiological experiments suggests at once that there must be some change in irritability, while the stimulating centre continues to work rhythmically. To explain this consistently, Hoffmann suggests that higher up in the vein than where the normal impulse takes its start there may be muscle fibres in which is generated a rhythm faster than the normal heart rate. If these, for instance, develop 4-N impulses, and, as in Kries' experiment, the neighboring fibres are able to respond to only every second, these will take up a 2-N rhythm, and the point where the normal cardiac contraction has its origin, able to answer only every other impulse, will take up a 1-N rhythm. If, then, the conductibility, irritability, and contractibility of the heart and of the fibres at this point be increased, it may come to pass that instead of every other impulse, each individual impulse from the point above may take effect, resulting in a doubling of the heart's rate. Similar conditions occurring higher up would then allow a quadruple heart action. For the origin of such influences Hoffmann looks to the nervous system.

Lommel cites a case which corroborates Hoffmann's observations, and accepts his deductions almost completely. He lays special emphasis on the pulsus alternans character of the curves at the beginning and end of an attack. Hoffmann in 1900 had suggested the view that attacks of paroxysmal tachycardia depended upon a heaping of extrasystoles, the continuous irritation giving rise to premature systoles as soon as the bounds of the refractory period were reached. The perfectly regular rhythmical recurrence of the pulse waves, the absence of any compensatory pause over a long period of time, and the curious proportion of increase in rate lead both Hoffmann and Lommel to discard this view. Gerhardt, however, has something to say in its favor. His curves and interpretations cannot be reproduced, but he

[1] Influences involving the development of the stimulus.

believes he has shown that a doubling of the pulse rate, as well as a gradual increase in the size of the interpolated wave (as in Hoffmann's curves at the onset of an attack), may be produced by extrasystoles. He lays emphasis upon the tendency to the production of extrasystoles just before and after an attack of paroxysmal tachycardia.

SURGERY.

UNDER THE CHARGE OF

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Scopolamine-morphine as an Adjuvant in the Administration of General Anæsthesia.—SEELIG (*Annals of Surgery*, August, 1905) presents a series of 65 general anæsthesia administrations by this method to show that it merits recommendation and trial. The operations invaded the abdomen, cranium, neck, and rectum. In every instance the patient received a hypodermic injection of scopolamine hydrobromate gr. $\frac{1}{100}$, of morphine gr. $\frac{1}{6}$, one-half hour before the administration of the general anæsthetic, which was induced by the ethyl chloride-ether sequence. The Bennet inhaler, with a slight modification for the administration of the ethyl chloride, was employed for the general anæsthesia.

Scopolamine is an alkaloid from the hyoscyamus niger. Its sister alkaloids are hyoscine, daturine, duboisine, atropine, and hyoscyamine. By some authorities it is claimed that scopolamine is closely allied to hyoscine; by others that the two are identical, while Ladenburg claims that hyoscine and scopolamine are by no means identical.

Kochman states that there has never been a death from scopolamine. By the method of administration and dosage worked out by Korff, three doses are given, at three, one, and one-half hours before operation. Rational objections have been made to the fixed dosage. Three deaths have occurred.

Seelig is enthusiastic in its praise. Of his 65 cases, only 1 vomited or retched while on the operating table; 77 per cent. did not vomit at all. One-third of the cases that did vomit, vomited only once, and then only from 2 drachms to 1 ounce of clear mucus. Nausea was never pronounced, except in 2 cases, and vomiting never occurred earlier than two hours after the operation. Laryngeal sensibility was intact by that time and acted as a complete safeguard against tracheal inspiration, and the consequent danger of aspiration pneumonia. Besides decreasing the tendency to vomit, it lessened the quantity of the anæsthetic necessary, only 4 ounces per hour at operation being required.

By the administration of scopolamine-morphine the patients go under and come out from the influence of the general anæsthetic without passing through the usual stages of excitement. Salivation is almost invariably absent. Much less pain is experienced by the patient in the first twenty-four hours after operation. These advantages have been confirmed by Tuffier, Israel, Dork, and Robertson (Robertson used hyoscine instead of scopolamine). Morphine lessens the afferent nerve impulses, reaching the vasomotor centres, and thereby lessens shock (Crile). Scopolamine raises the blood pressure and thereby aids in preventing shock. The consensus of opinion is that many of the irregularities in the action of scopolamine are referable to impure preparations of the drug. Merck's scopolamine is the best preparation of the drug.

Scopolamine-morphine Anæsthesia.—Ries (*Annals of Surgery*, August, 1905) says that the greatest difficulty has been found in determining the best proportion of the two drugs to employ. Too much morphine will affect the heart dangerously, while too much scopolamine produces a state of excitement similar to that following atropine poisoning. The patients become restless and talk as if in a delirium. The pupils are widely dilated and rigid and the face is flushed. They demand water constantly and complain of dryness; but the dryness soon wears off, and neither in man nor in dogs have any cases become known where scopolamine alone has produced death.

Scopolamine-morphine is used for two purposes: First, with the intention of performing the whole operation under this anæsthesia alone; secondly, as a preliminary to anæsthesia by inhalation of chloroform or ether. If the case is a complete success the patient becomes sleepy after the first injection, is fast asleep after the second, and is unconscious and insensible to pain after the third. The operation may begin one-half hour after the last injection.

According to Ries' experience, the sleep may be so slight that the patient awakes when spoken to or moved. Some, if aroused, cannot answer intelligently, but mumble a few unconnected words. Others again, though they cannot be aroused, move when being handled, or even complain. It then becomes necessary to add some other anæsthetic to the scopolamine-morphine. Ries has, in a number of cases, added infiltration anæsthesia with a 0.06 per cent. salt solution without cocaine, to the complete exclusion of chloroform. When chloroform is given very little is required, and in many cases after the first few drops have been given for the first incision no more chloroform is necessary, so that operations lasting fifteen to thirty minutes can be finished with less than a teaspoonful of chloroform. Complete muscular relaxation does not, as a rule, result.

Ries has used scopolamine-morphine on 72 patients in 92 operations. Of the 72 patients, 3 died. In none could the scopolamine-morphine be accused. Of the deaths reported as due to it, only 1, in Ries' opinion, seemed to be due to the scopolamine-morphine. He has used hyoscine instead of the scopolamine, and with both had the same result.

Scopolamine, when dissolved in water, does not keep long, and it has been Ries' rule to have a new solution made every week. Even then it was noticed that the effect of the drug would decrease toward the end of the week, so that in the future he will have a new solution made every three days.

The Relationships between Colitis and Appendicitis from a Surgical Point of View.—Lockwood (*British Medical Journal*, March 4, 1905, p. 466) says that any form of colitis may be associated with appendicitis, and that it is of the very highest importance that we should try to learn their relations to each other. Without doubt appendicitis can cause colitis. In many operations for the removal of the appendix the end of the ileum, the cæcum, and the right colon are obviously inflamed. In addition there is tenderness along the course of the right colon and rigidity of the abdominal wall. Mucus or mucus tinged with blood in the stools has been associated, showing that inflammation on the outside of the colon must also have involved the mucous coat.

The relationship between mucous colitis and appendicitis is a mere question of extension by proximity, facilitated, perhaps, by identity of vascular supply. In cases of this kind the bowels soon recover when the appendicitis has ceased. The appendicitis is the dominant note.

Colitis may be the prominent feature to such an extent that the appendicitis may be obscured and overlooked.

Two cases are referred to of appendicitis with left-sided pain, but with absence of pain and tenderness over the appendix. The cause of the left-sided pain was clearly colitis. In both the inflamed appendix was hidden behind the right colon, which might have afforded some sort of protection to it. The writer says that while he does not know that appendicitis can cause the mucomembranous form of colitis, he does know that mucomembranous colitis can cause appendicitis. In the last case referred to in his article, postmortem examination showed the whole depth of the mucosa ulcerated away, so that no trace of the tubular glands remained. The submucous, muscular, and peritoneal coats were much inflamed, with dilatation of their bloodvessels and lymphatics. There had been no reason to suspect that the appendix had suffered, but microscopically it was ulcerated exactly the same as the intestines.

On Ascending Currents in Mucous Canals and Gland Ducts and Their Influence on Infection.—Bond (*British Medical Journal*, July 29, 1905, p. 232) says that by some means or other and under certain conditions particles of an insoluble substance, such as indigo, inserted into the orifice of a mucous canal or duct are conveyed along the mucous channel in a reverse direction to that taken by the contents of the tube or by the secretion or excretion of the glands along the ducts. The conditions which seem to favor this passage are: First, some interference with the normal flow of the contents of the mucous tube or duct; some arrest or diversion of secretion, such as is produced by a fistulous opening, though it is by no means necessary that this should be complete.

That this movement is not due to ciliary action is shown by the fact that it occurred where cilia are absent. While irregular or reversed peristalsis might cause it, it is suggestive that the ascent of the indigo up the colon was not prevented but rather accelerated by the increased downward peristalsis produced by purgatives. Reversed muscular contractions are as yet, moreover, unknown or rather undemonstrated in the bile-ducts, urethra, and urinary tubes, Fallopian tubes, or in the ducts or glands, in all of which the reversed mucous currents were demonstrated. It is not due to a physical agency alone, such as capillary action. This is absent in the non-living tube.

The essential conditions seem to be a living tube, whose walls are

partially if not wholly in apposition and lined by a mucous secretion; while the reversal of this mucous current is favored by any condition (such as a fistula) which leads to arrest or diversion of the ordinary secretion of the viscus or gland.

The bearing of these remarks on the larger question of infection will be at once apparent. Bond is inclined to think that in some cases more attention should be paid to mucous channels as routes of infection rather than to the lymph or blood stream. Bearing in mind the proved transference of particles under certain conditions from duodenum to gall-bladder and from urethra to ureter, it is to be hoped that further light may thus be thrown on these calculous diseases.

THERAPEUTICS.

UNDER THE CHARGE OF

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The Treatment of Heart Affections.—Dr. E. de Renzi, from a prophylactic standpoint, advocates the administration of large doses of sodium salicylate (1½ to 2 drachms per day) in all cases of acute articular rheumatism and the proper treatment of all gouty tendencies and of obesity. In endocarditis with compensation, hygienic treatment is necessary, but digitalis is contraindicated. On the other hand, lack of compensation necessitates reliance upon this drug, the best preparation of which is, in the author's opinion, soluble digitoxin. Its action takes place quickly and it presents no disadvantages. Lesions of the heart muscle may be benefited by exercise, under the influence of which the myocardium becomes strengthened and hypertrophies. In this connection, Oertel's method increases the respiratory power, accelerates the elimination of toxins and facilitates the peripheral circulation. It is specially useful in fat patients and in functional heart lesions. Massage and Swedish movements lessen the peripheral resistance, dilate the capillaries, and militate against venous congestion. These are indicated in organic lesions.—*Berliner klinische Wochenschrift*, 1905, No. 11, p. 291.

The X-ray Treatment of Tineæ.—Dr. Dupeyrac reports the results of the treatment of a case of tinea tonsurans, a case of *trichophytosis barbæ*, and a case of favus of the scalp. These were well treated by an *x*-ray of slight penetration, and the surrounding parts were shielded by a protection of lead. In the first case epilation was complete on the twentieth day and a cure was achieved in three and one-half months; in the second case treatment was continued for a month, and two months

later the cure was complete. The third patient is still under treatment, but the ray does not seem to be in this instance superior to the forceps for purposes of epilation. The author considers that as he has employed them, the rays do not act as a parasiticide but only as a depilatory agent, thus making it possible to thoroughly clean the site of the lesion. There is difficulty in regulating the strength of the ray, so that it shall exert this depilatory action and yet not produce a dermatitis and complete alopecia, consequently he employs rays of slight penetration and of an amount corresponding to 5 units H.—*Marseille médicale*, 1905, No. 6, p. 173.

The Treatment of Gastric and Intestinal Hemorrhage.—Dr. Capitan considers calcium chloride the most useful drug in these conditions. In typhoid hemorrhage the baths are to be stopped, the patient is kept as quiet as possible; milk is replaced for three days by a fixed quantity of water. Two intestinal irrigations per day of a quart of water each at 118°, to which a drachm of calcium chloride has been added, are given. At the same time the patient is given 30 grains of this substance in aqueous solution by mouth. The irrigation must be given gently, slowly, and under low pressure. Four or five small doses of opium by mouth complete the treatment. Gastric hemorrhage is to be managed in practically the same manner. The intestinal irrigations may be supplemented by injections of serum and other means calculated to sustain the patient should be employed. The irrigations relieve the intestine of the extravasated blood, and, given at a temperature of 118°, they have both a constricting effect upon the bloodvessels and act as a nerve sedative. The calcium chloride increases the coagulability of the blood and enhances the hæmostatic effect of the hot water. This agent is not toxic, is not disturbing to the stomach, and is readily absorbed. Its action takes place in about fifteen minutes, but taken in larger doses than those above mentioned, it loses its power of increasing blood coagulability.—*La médecine moderne*, 1905, No. 10, p. 76.

Dechloridation in Epilepsy.—Dr. Arthur Morton reports encouraging results from the substitution of sodium bromide for sodium chloride in the food of epileptics. The sodium chloride was not wholly excluded from the diet, but enough of the bromide was added to the food so that each patient took about 15 grains per day. It is claimed that this salt is rapidly absorbed and becomes part of the body tissue when given in this manner and that only about half the usual quantity is necessary to produce the sedative effect. The author concludes that this method controls the convulsions and has little if any affect upon the nutrition. It is apt to cause constipation and does not furnish enough salt to satisfy the patient. It may be advantageously employed in intelligent patients, but is useless in middle-grade epileptics, as they have neither the desire nor the will to carry it out. A modified salt-poor diet, in which about equal parts of sodium chloride and bromide are used in the food, may be employed with advantage with idiotic and demented patients if their diet can be absolutely controlled. Bromism is comparatively rare.—*Boston Medical and Surgical Journal*, 1905, No. 24, p. 698.

Antistreptococcus Serum in Scarlatina.—Dr. L. Mendelsohn finds that the serum is very prone to produce eruptions in scarlet fever patients

and with the exanthem at times a slight degree of albuminuria appears. The injections are contraindicated when heart lesions are present. To produce its best effects, the serum should be given during the first week of the disease, and even then its influence upon the temperature and the eruption is slight. It does, however, favorably affect the angina, and severe throat complications are rare under its use; glandular enlargements seem to be less frequent in injected cases. The author gives the impression that he considers the employment of the serum of rather doubtful value.—*Deutsche medizinische Wochenschrift*, 1905, No. 12, p. 461.

The Treatment of Scarlatina.—Dr. E. Ausset discusses the treatment of scarlatina under three headings: (1) To combat the pyresis and sustain the organism. The temperature should be controlled by baths, beginning at 82°, and, if necessary, lowered to 68°. So low a temperature as the latter should never be employed unless a good reaction is obtained. The bath acts as a heart sedative and a nerve tonic as well as an antipyretic, and it is ill-advised to attempt great reduction of temperature by this means. Antipyretic drugs, save possibly cryogenine, should not be given. To sustain the system tonics, such as ammonium acetate, kola, or canella, are indicated. (2) To eliminate the toxins. Fluid diet and the baths assist diuresis, and this should be further augmented by the ingestion of plenty of fluid. Theobromine, 30 grains per day, is an excellent drug in this connection. Intestinal irrigations of saline solution, in the author's opinion, may produce albuminuria and œdema. The bowels should be kept freely open. The heart action should be watched, and if a tonic is necessary, sparteine sulphate, 0.15 to 0.3 grain, may be given hypodermically. Digitalis is toxic and caffeine excitant in children. (3) To stimulate the nervous system: alcohol in the form of champagne may be given in small doses and hypodermatic injections of camphor in oil may be employed. Heart failure is to be treated by baths at 90° to 95° and oxygen inhalations.—*Annales de la policlinique de Paris*, 1905, No. 4, p. 80.

Viferral, a New Hypnotic.—Drs. K. Witthauer and S. Gaertner report this combination of anhydrous chloral and pyridine. It is a white powder, slowly soluble in cold water, easily soluble in hot. It has a bitter taste, and is less toxic than chloral hydrate, does not disturb the digestion, nor depress the heart. In ordinary dose (15 grains), it causes a sound, restful sleep, usually without unpleasant after-effects. Rarely there may be slight headache. If given for considerable periods, a tolerance may become established.—*Thérapeutische Monatshefte*, 1905, No. 3, p. 143.

Electric and Hydrotherapeutic Treatment in Heart Affections.—Dr. G. Zimmermann finds that electric baths with the alternating or sinusoidal current may be employed advantageously in cardiac affections; the palpitation and dyspnœa are relieved, the appetite is increased, and the patient's sleep better; the pulse becomes slower, the œdema less, and the blood pressure higher. In cardiac hypertrophy, however, baths with the alternating current are contraindicated; here the iodides or the nitrites should be given to lessen the peripheral resistance. Baths with the alternating current act well in dilatation resulting from an atonic

myocardium, and in emphysema, but not unless the heart muscle is able to regenerate itself by compensatory hypertrophy. The author believes that these baths are chiefly useful in circulatory disturbances with low blood pressure, early failing compensation, moderate degrees of fatty degeneration, atonic conditions of the heart muscle and arterial system, and in pulmonary emphysema. While in sclerotic conditions the Nauheim treatment is more reliable.—*Münchener medizinische Wochenschrift*, 1905, No. 12, p. 549.

OBSTETRICS.

UNDER THE CHARGE OF

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Complications in Labor Caused by Anomalies of the Amnion.—Bardeleben (*Zeitschrift für Geburtshülfe u. Gynäkologie*, 1905, Band liv., Heft 2) draws attention to the complications which may result from adhesions of the amnion to the fetus.

In 2 cases of young primiparæ the uterus remained between pains so hard and firmly contracted as to occasion suspicion of uterine tetanus. On vaginal examination it was difficult to find the external os. The head was movable. The membrane seemed unusually hard and resisting, and when finally ruptured but very little amniotic fluid escaped. In 1 case spontaneous birth followed, and in the other delivery by forceps. Mothers and children recovered without interruption. In 1 case the placenta and membranes showed nothing abnormal, but the small size of the membranes; in the other the shape of the placenta was abnormal, the umbilical cord very slightly coiled, the chorion easily separated from the amnion, but firmly joined to the decidua; the amniotic cavity could contain only a fraction of the usual quantity of amniotic liquid.

The writer also reports 2 cases where the amnion had caused direct interference with the fetus. In 1 at the fifth month of pregnancy there was prolapse of the umbilical cord, the patient having had hydrorrhœa for two months before. The fetus developed outside the membranes for three months. The umbilical cord was disproportionately long and reached to the vulva. Occlusion of the cord caused fetal death and made it necessary to empty the uterus. The cervix was found unusually resisting; the uterus did not promptly contract, and profuse bleeding occurred.

In the second case the patient was thought to have placenta prævia; she was, however, in good condition and had not had sudden and severe hemorrhage. It was thought that the placenta had become entirely loosened and that the presenting part was acting as a tampon. On

examination the fetal head could be felt and in front of it over a great extent of the head a sponge-like mass. There was a cerebral hernia, and the midwife in examining had pierced the brain substance, which caused bleeding. A broad amniotic band extended from the sac of the cerebral hernia to the back of the fetus, to the placenta and also to the region of the left hand. This band ruptured when strong pains occurred and the child was delivered. The membranes were completely developed and easily separated from each other. In this case the amniotic band had prevented the descent of the child, while the abnormality in the cranium and the perforation of the brain had produced hemorrhage.

He further describes a case in which the fetus was outside the amnion, in which the membranes had ruptured and hemorrhage had occurred. On vaginal examination the cervix was found dilated and filled with fetal parts, which with difficulty could be distinguished. It was finally made out that there was a monstrosity with abnormalities in the membranes and a very large placenta. The umbilical cord was very short, but 14 cm.; as the fetus descended traction upon the cord loosened the placenta.

That such a complication can produce a fatal issue is illustrated by a case in which a multipara having a breech presentation and fully dilated cervix had labor cease after seven hours' duration. On examination an oblique transverse presentation was present, with an abnormality in the membranes, and also a rupture of the uterus. The left shoulder of the child had torn its way through the uterine wall; it was possible by very gentle traction to deliver a malformed fetus, placenta, and membranes. A thick amniotic band had caused the elbow to be strongly bent and held it in that condition. A similar condition was present in one of the knees. After delivery the rent was tamponed with gauze in the hope that the patient might recover. Fever and renewed bleeding occurred, and the patient perished on the ninth day from fatal hemorrhage. On autopsy a large hæmatoma was found on the left side extending to the kidney. In the wound in the perimetrium was a large-sized vessel, which was not completely thrombosed and from which came the fatal bleeding.

Disturbances of Digestion in the First Days of Life.—In the *Archiv für Gynäkologie*, 1905, Band lxxv., Heft 2, KERMAUNER contributes an interesting paper upon the disturbances of digestion during the first days of the infant life.

The high mortality of the first year of life has been repeatedly demonstrated. From 22 to 25 per cent. is a moderate estimate of the number of infantile deaths during the first year. One-third of these arise from some disease in the digestive organs. From 70 to 80 per cent. of those infants dying during the first year had some disturbance of digestion. Furthermore, the greatest mortality among infants is in the first two months of life, diminishing steadily after that time. Kermauner believes that this arises through errors in the care of newborn children. In studying this subject, he examined the discharges from the intestines of the infants under hospital care and found abundant germs in the matter discharged from the intestines; this was independent of cases of infection arising among the mothers. In some cases the mother's milk contained staphylococci. He succeeded in reducing the morbidity and the mortality in the Heidelberg clinic by two procedures. The first was

a change in the manner of nursing these children, whereby an increased number of trained nurses was provided, and minute attention given to the atmosphere of the wards, to disinfection of diapers, the care of the umbilicus, the use of sterile powder, of sterile water for bathing, and also of soap. The closest attention was given to all the details of asepsis and sufficient nurses were employed to carry this out.

A second factor of improvement was the prevention of loss of heat on the part of the children; care was taken to keep the children properly clothed and to avoid exposure of the child at the time of bathing. The clothing of the children was not changed. A further precaution was the removal of the children from the bed of the mother; each child was given a small but separate crib, and contamination from one child to another was carefully avoided.

The results of this treatment were exceedingly good and demonstrated clearly the correctness of the reasoning which led to its institution.

Virulence of Streptococci Found in the Vagina of Pregnant Patients.—WALTHARD (*Zeitschrift für Geburtshülfe und Gynäkologie*, 1905, Band liv., Heft 2) has studied this subject by observing the agglutination of streptococci obtained from pregnant patients. Clinically speaking, he is unable to differentiate these germs from those of less degree of virulence; and his investigations show that streptococci remaining in the vagina of pregnant patients for a considerable time where the patient is not interfered with, so far as the patient is concerned, are practically non-virulent saprophytes.

Gangrene after Eclampsia.—In the *Monatsschrift für Geburtshülfe und Gynäkologie*, 1905, Band xxi., Heft 6, GUTBROD describes two cases of gangrene occurring after eclampsia.

The first was that of a primipara, aged twenty-two years, who had severe eclamptic convulsions and remained unconscious after the delivery of the child. She was treated by hot packs and perspired freely. In her unconscious condition she lay much of the time with the left hand under the back and the left foot crossed over the right. As she began to recover from eclampsia, gangrene of the left hand developed, while the skin over the sacrum against which the hand had pressed became also gangrenous. The same condition developed on the inner side of both knee-joints, on the under side of the left foot, and on the dorsal surface of the right foot. Forty-six days after the beginning of her illness the patient had improved considerably, but the tendons on the right foot had been exposed by slough, suppuration burrowed into the foot, and it was finally necessary to amputate it.

The second case was that of a multipara who had eclampsia with spontaneous expulsion of the child. After labor the eclampsia persisted; the patient had profuse bronchial catarrh, with threatened œdema of the lungs, which gradually subsided. She remained unconscious for some time after delivery, and on the fifth day after confinement the great toe of the left foot began to turn black. The patient had fever, and the entire foot became deeply red. Her convalescence was very slow, as pus formed which required incisions in several parts of the body, and the cornea became clouded. Over four months was consumed in the patient's convalescence. She finally made a good recovery.

GYNECOLOGY.

UNDER THE CHARGE OF

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Periappendicitis Decidualis under This Term.—HIRSCHBERG (*Zentralblatt f. Gynäkologie*, 1905, No. 28) describes a peculiar condition seen in an appendix which was found to be adherent to a tubal mole. Microscopic examination showed that in addition to the hypertrophy of the peritoneal endothelium due to the irritation of the effused blood, there was an actual growth of decidual cells, while in the serosa itself were isolated groups of such cells. Schmorl has shown that even in normal pregnancy the cells of the peritoneum may undergo a similar change, as shown by the "decidual reaction," or the homogeneous staining of the protoplasm. The same writer states that while such cells are found in the non-adherent omentum, they may be absent when it is attached to the uterus.

Report on 1000 Laparotomies.—VON PODHARITZKY (*Archiv f. Gynäkologie*, Band lxxiv., Heft 3) reports a series of 1000 abdominal sections performed by Fenomenoff in the course of seventeen years. No antiseptic solutions were used during the operations, the hands and field of operation being cleansed according to Mikulicz's method with tincture of gum soap, alcohol, and tincture of iodine. The suture material was silk boiled in sublimate solution. There were 500 ovariotomies and 280 supravaginal amputations, 60 salpingo-oöphorectomies, and 58 cases of ectopic gestation; in addition to this, Cæsarean section was performed six times and the abdomen was opened seven times for rupture of the uterus. The combined mortality was only 5.1 per cent.

Calcification of the Fimbriæ of the Tubes.—WAGNER (*Archiv f. Gynäkologie*, Band lxxiv., Heft 3) describes the following condition found at autopsy: The fimbriæ of the tubes were deeply congested and yellowish-white in color and were covered with small granules. Under the microscope the latter were found to be small calcareous nodules in the tissue of the fimbriæ, which were swollen and their vessels congested. In a few places the intima was thickened and calcareous. No nodules were present in the connective-tissue spaces. The writer refers to a similar case described by Schmitt, in which the nodules were referred to persistent circulatory disturbance in consequence of cardiac insufficiency.

Operation for Prolapse of the Rectum.—WENZEL (*Deutsche Zeitschrift f. Chirurgie*, Band lxxvi., Heft 1) refers to the older methods of treating rectal prolapse with the cautery, ligature, and the more modern procedures of resection and suspension. He believes that in many cases the cause is due to the unusual depth of Douglas' pouch, and that the object

of the operation must be to fold in the peritoneal pouch, as well as to suture the relaxed muscles of the pelvic floor and external sphincter. In one instance after opening the abdomen he first suspended the retroflexed uterus, then dissected off the peritoneum from the uterus, vagina, and rectum, and sutured the rectum to the posterior surface of the uterus and vagina, fixing the sigmoid lateral and in front to the abdominal wall in order to prevent a kink in the gut. The operation was successful, but the patient was obstinately constipated and the prolapse gradually recurred. The writer thinks that the latter might have been avoided by simultaneously contracting the sphincter.

Resection of the Bladder for Cancer.—GOLDENBERG (*Beiträge zur klin. Chirurgie*, Band xliv., Heft 3), in reporting a case, refers to the 57 other reported cases, in which the greatest trouble was experienced in managing the ureters. Implantation in the rectum is not admissible, on account of the danger of ascending infection; on the other hand, the trigone is so often diseased that they cannot be sutured here. The writer has experimented with dogs with the view of utilizing the lower portion of the ileum for this purpose, a coil of gut being sutured to the abdominal wall and opened. When the ureters have become firmly attached to the gut the intestinal fistula can be closed.

Intermenstrual Pains.—VAN DE VELDE (*Zentralblatt f. Gynäkologie*, 1905, No. 30) believes that the periodical variations in the menstrual blood pressure are due to some chemical irritation proceeding from the ovaries. From observation of various blood curves and experiments with ovarian tablets, he infers that the menstrual flow is due to a falling of the pressure or general diminution of the vascular tone. Patients with a high blood pressure are apt to have so-called intermenstrual pains, which he explains as due to passive hyperæmia of the uterus. These are noticeably relieved by the administration of ovarian extract, which modify metabolism so that the wave sinks.

Double Ovariotomy during Pregnancy.—HEINSIUS (*Zentralblatt f. Gynäkologie*, 1905, No. 32) reports the case of a five months' pregnant woman with severe abdominal pain and ascites. A movable pedunculated tumor the size of two fists was found in the right iliac region. On opening the abdomen this proved to be an ovarian dermoid with a complete twist of the pedicle and firm intestinal adhesions. A second dermoid was removed on the left side, thus being a torsion of 180 degrees. Recovery without interruption of the pregnancy.

Sexual Irritation in Early Tuberculosis.—LANDRET (*Zentralblatt f. Gynäkologie*, 1905, No. 32) calls attention to the fact that in the early stage of tuberculosis there may be increased sexual desire and capacity for procreation. He attributes this to the presence of the tuberculous toxin in the system, and thinks that it may prove valuable as an early diagnostic symptom.

Free Bodies in the Tubes.—FEDOROW (*Ann. de gyn. et d'obstétrique; Zentralblatt f. Gynäkologie*, 1905, No. 32) found in a sactosalpinx fifteen

free nodules varying in size from a pea to a bean, of different shapes, round, oval, and polygonal. Over the surface of the latter were scattered small villi. The bodies were soft, encapsulated, and microscopically presented the appearance of adenoma, with small cavities containing papillomatous masses. When the fibrous capsule was thick the interior of the body showed marked degeneration, containing only empty, atrophied bloodvessels, and necrotic epithelium. The influence was that there were papillomata that had become detached from the living membrane of the tube.

OPHTHALMOLOGY.

UNDER THE CHARGE OF

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Treatment of Severe Blepharospasm by Artificial Anastomosis of the Facial with the Spinal Accessory Nerve.—ABADIE et CUNEO, Paris (*Arch. d'Oph.*, April, 1905), have successfully practised section of the facial nerve, followed by immediate anastomosis with the eleventh pair in a severe case of this affection, which had been rebellious to both medical and surgical attempts at cure. Section of the facial nerve is followed by a temporary paralysis; the latter soon recovers.

Aural Nystagmus.—BARANY (address before the Vienna Ophthalmological Society, *Centralblatt f. Augenh.*, August, 1905) was able to excite nystagmus by syringing the external auditory passages with water. He experimented upon 100 individuals, including some whose hearing was normal, some with aural disease, and some were deaf mutes. His results were as follows:

1. If the temperature of the water injected was below that of the body an almost purely rotatory nystagmus was excited directed to the opposite side of the body; the phenomenon was most marked as the patient looked toward the side opposite that of the ear which was being treated. If the injected water was above the body temperature an almost purely rotatory nystagmus was also excited, but it was directed toward the side of the ear which was under treatment and most marked as the patient looked in that direction. These phenomena only occurred with the head erect.

When the patient's head was inclined toward the side not under treatment there occurred in the first case (water below body temperature) a horizontal nystagmus directed toward the side under treatment; in the second case (water above body temperature) toward the side not

treated; this gave way to the above-mentioned rotatory nystagmus only as the head was erected or inclined toward the side under treatment.

2. If the injected water be of the temperature of the body no nystagmus occurs.

3. The nystagmus mentioned under 1 may be excited from either ear, the reflex paths being intact; nevertheless longer time and greater temperature differences are required where the drum membrane is present. Where the wall of the labyrinth is exposed the nystagmus occurs with very small temperature differences and at the expiration of five to ten seconds.

4. If the reflex path has been interrupted nystagmus cannot be excited from the affected ear, but it may be produced as above described by acting upon the other ear if it is sound.

5. The nystagmus is usually accompanied by vertigo and disturbance of equilibrium, occasionally also by nausea, faintness, and vomiting. All of these symptoms may be absent.

Where the disturbance of equilibrium is marked the subjects fall to the ground; the falling occurs in a certain definite direction; it agrees in general with that observed in disease of the vestibular apparatus.

As to the cause of these phenomena, the speaker regarded it as probable that the warming and cooling of the contents of the labyrinth acted as an irritant upon the terminals of the nerves. It has been noted that temperature stimuli may excite attacks in case of Meniere's syndrome.

Is Trachoma Contagious?—This question is hardly worth while discussing. Still, as there appear to be a few ophthalmologists who have some doubt upon the subject, it may be worth while to put on record an instance as typical as the one reported by POULARD (*Arch. d'ophthal.*, August, 1905), where all the members of a family and others who were brought into intimate contact with them became successively affected with the one disease.

The other inhabitants of the tenement in which the family resided were convinced of the danger of contagion and finally succeeded in having the family expelled, because, following their expression, "they were giving sore eyes to the whole house."

Insufficiency of Convergence.—LANDOLT (*Arch. d'ophthal.*, July, 1905) reports a case of this condition which is remarkable from the completeness of the result obtained by an operative procedure, of a kind which on first thought would hardly seem capable of leading to that result.

A girl, aged thirteen years, suffered from a rebellious asthenopia, which had long disabled her from doing fine work in a sewing establishment where she was employed. The left eye showed myopic astigmatism 0.50 D.; the right was emmetropic. The vision in each eye equalled 1.25. The accommodation was normal in both eyes. The cause of the complaints was found in defective convergence, which, indeed, was highly faulty. The maximum of this function was only 2.5 metre angles. The minimum of convergence, which is normally negative, was positive and corresponded to +1.75 metre angles, so that the patient could not get the visual lines parallel, the punctum remotum being situated at —0.507 in front of her. There was thus

present besides the insufficiency of convergence, that is to say, relative divergent strabismus, a convergent strabismus of 5.8. There existed in fact crossed diplopia inside of 40 cm. and homonymous diplopia beyond 50.

The history explains this strange condition. The child previously had had a tenotomy of the right externus performed, no doubt with a view of correcting the insufficiency of convergence, under the fatal error that weakening the external recti should strengthen the power of the interni. The operation, which was quite a moderate one, had for its effect to narrow the field of excursion of that eye. While the excursion outward of the left eye equalled 58 degrees, it was only 40 degrees in the right eye. The movements inward were 44 degrees right and 42 degrees for the left eye. This apparent gain of 2 degrees cannot be certainly set down as due to the operation, because the adductive power before the operation is not known. If it be admitted that this was equal in the two eyes, this increase of 2 degrees would have been paid for by a sacrifice of 18 degrees of adductive power.

In any case the tenotomy of the right externus had the effect of transforming the facultative divergence into a convergent strabismus with homonymous diplopia, while leaving a considerable insufficiency of convergence.

The condition was very embarrassing. Theoretically, it seemed necessary to detach the muscle unfortunately set back and reattach it to its normal insertion and to advance one of the internal recti to remedy the insufficiency of convergence. It was finally decided to attempt the re-establishment of the power of positive convergence by advancing one of the interni. The left was selected. Improvement was rapid, the amplitude of convergence increased in both directions, the punctum remotum receding while the punctum proximum approached the eyes. Ten days after the operation the minimum of the convergence was 0.75, the maximum 9 metre angles at the end of two months, the minimum had become negative, —0.50, while the maximum was 10 metre angles, that is to say, quite normal. A month later the conditions were still more favorable, having indeed surpassed the normal at both extremities, extending from —1.75 to 12 metre angles.

The advancement had thus resulted in a gain of 13 metre angles, divided between 9.5 in the direction of the punctum proximum and 3.5 toward that of the punctum remotum.

It is very remarkable that the advancement of the internus, while so greatly augmenting the power of convergence, did not also increase the convergent strabismus. On the contrary, this strabismus slowly disappeared and even gave place to a marked facultative divergence. The result obtained in this case, if we base our views upon the old but erroneous theory of the influence of muscle operations upon the movements of the eyes, is incomprehensible. It is explicable upon the view that tenotomy, that is to say, setting back of a rectus, causes the eye to advance outward, so to speak, from its muscular funnel and tend to lose excursive power, not only in the direction of the muscle which has been set back, but even on the opposite side. By advancement, on the contrary, we sink the eye more deeply into this funnel and give the muscles greater influence upon the rotation of the globe leading to increased power of all the muscles affecting the rotation of the eyeballs.

[This case is certainly remarkable. Were it not for the eminence and acknowledged skill of the reporter, one might be inclined to suspect that a neurotic element may have been present and that hysteria and suggestion entered into the symptoms and the cure.—ED.]

OTOLOGY.

UNDER THE CHARGE OF

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Regional Anæsthesia of the External Auditory Canal.—P. LAVAL (*Archiv f. Ohrenheilkunde*, Band lxiv., Heft 2 and 3), on the recently published experiments of von Eiken on local anæsthesia of the external auditory canal, with secondary anæsthetic effect upon the middle ear permitting removal of the ossicles, has led the writer to publish his own experiments, made by a similar method, independent of those of von Eiken's in the past summer in this field of work and since successively repeated.

Since the previously proposed methods of local anæsthesia had proved inadequate for special reasons, the Schleich infiltration anæsthesia limiting the space anæsthetized by wheal formation, the ethyl chloride anæsthesia being hardly applicable on account of the severe after-pains, and the higher per cent. solutions of cocaine having proved ineffectual, the writer adopted the method of regional anæsthesia by injections along the course of the nerves supplying the external auditory canal, the procedure being, therefore, an application of Oberst's original method for anæsthetizing fingers and toes, developed in later years by Braun.

The field of operation in the external canal and middle ear is supplied by three nerve trunks with sensory fibres, whose anatomical relationships were determined by successive comparative dissections in three cadavers.

Anteriorly the nervous meatus accustici externi enters a branch of the auriculotemporalis with two or three fibres to the external auditory canal, then it breaks through the fibrocartilaginous part close to the lateral part of the osseous canal, in order to ramify inward and outward in the fibrocartilaginous canal. Its outer limits of ramification lie close behind the crest of the tragus, inward it takes part in the innervation of the drum membrane. Similarly situated is the ramus auricularis vagi, which passes through the canaliculus mastoideus laterally from the os timpanicum to the fibromembranous canal and divides into two branches. It supplies the posterior wall, the greater part of the drum membrane, and a zone about 0.5 cm. broad and applies itself half-moon shaped in the cavum conchæ to the entrance of the external auditory canal. The

third nerve is a branch of the nervous auricularis magnus, which ascends with greater regularity from below to the sternocleidomastoid muscle, running subcutaneously in the axis of the antitragus to the posterior surface of the concha, and there dividing into several branches, distributing itself in the region of the cauda helicis to the lateral surface of the auricle. It supplies a part of the concha, lying below a line drawn horizontally through the tragus, as well as the lobulus, with sensory fibres, but on the posterior surface the line lies about 1 cm. lower.

In the greater number of operations, in the region of the external auditory canal, only the first two named nerves come into consideration, though there were cases in which a painless operation could be assured only by anæsthetizing the third nerve also.

In seeking the anterior branches it is possible to injure the temporal artery, the auriculotemporalis nerve, the capsule of the maxillary joint, and the parotid gland. However, if the mouth of the patient is opened as widely as possible the first two named organs sink deeper inward, together with their surrounding fatty tissue, as is easily demonstrable on the cadaver, and as can be tacitly appreciated by pressure in the groove in front of the tragus in the living subject, the folds of the capsule of the joint being tightly stretched and the parotid gland with the ascending maxillary branch being thrown forward. The facial nerve need not be considered, since it runs at the lower end of the external auditory canal, and the canal itself is avoided in the anterior as well as in the posterior injection by pulling the auricle upward and backward.

After disinfection and anæsthetizing the outer skin with a spray of ethyl chloride, the needle of a Pravaz syringe is inserted about 0.5 cm. in front of the tragus at the height of and in the direction of the floor of the external auditory canal perpendicularly to a depth of 1.5 cm., the injection fluid being made to flow during this process by constant pressure, the whole amount injected being 0.5 cm. The mouth should be widely opened and the auricle fixed by a gentle pull on the lobule. Where it is possible to open the mouth only partially rotation of the jaw toward the opposite articulation in the vertical axis will produce a compensating effect. The branch of the vagus is reached at the height of the canal by gently pulling the concha outward and forward and going in about 1 cm. in depth perpendicularly, close behind the fold of the auricle, between the cartilage and the bone of the mastoid process. From the same puncture opening, when necessary, the branch of the auriculus magnus can easily be reached, the needle, which is not completely removed from the puncture channel after injection, being pushed close under the skin about 1.5 cm. parallel to the posterior auricular fold forward and downward, and the final injection made into the subcutaneous adipose tissue close behind the lobule.

For all three injections 0.5 cm. of the solution is sufficient. Laval found that this quantity of fluid saturated the loose adipose and connective tissue through an area equivalent in extent to the size of a large cherry, and by bilateral injection of two cadavers with a weak watery solution of methylene blue, demonstrated that the nerve trunk was washed by the fluid to a great extent. In very fat individuals, in order to make sure, the dose should be increased to 0.75 cm.

The fluid used for injection in the clinical experiments was a solution of Braun's cocaine suprarenal tablets dissolved in 1 cm. of distilled

water. Each tablet contained cocaine muriate, 0.01; suprarenal borate, 0.00013, and sodium chlorate, 0.009, the result being a 1 per cent. solution of cocaine, the addition of 1 cm. of salt solution affording the equivalent of 0.5 per cent. solution of cocaine in physiological salt solution. In the first experiments a weaker solution only was used, but in the case of a strong thirty-five-year-old man, it did not diminish the sensibility, and the writer has since then favored the 1 per cent. solution for men, and for women and children the weaker 0.5 per cent. solution. The anæsthesia begins in about five minutes and lasts usually fifteen to twenty minutes; in one case, a girl, aged eighteen years, it lasted over three-quarters of an hour. In all cases the injection, as well as the operation of puncture, was painless and no evidences of cocaine intoxication were observed. As to after-pain, aside from slight sensitiveness of the place of injection for some time after the operation, there was no reported complaint on the part of any of the patients.

In the summer of 1902, 10 or 11 cases were operated upon under anæsthesia and 4 more in the following winter. The majority of the operations consisted in incision and curetting of furuncles in the canal. Of these, 1 is worthy of notice, a case in which a furuncle became an extensive abscess, completely closing the entrance to the canal and extending about 1 cm. downward in the cavum conchæ. By the third injection at the posterior point of puncture, the writer succeeded in anæsthetizing the lower half of the auricle, made a long incision, and curetted without causing pain. In 2 cases there were polypoid granulations about the size of peas, one from the edge of a deep ulcer, the other, with total defect of the drum membrane, resting on the posterior inferior part of the carious annulus tympanicus. A case of carcinoma of the canal in a man, aged about forty years, was of especial interest. The suspicious tumor, half the size of a pea, rested posteriorly and inferiorly on the lateral border of the middle third of the external auditory canal. The tumor was completely removed by an oval incision, including a strip of normal skin, then elevated from the bone and removed with forceps without causing pain. The result of the histological examination gave indications for radical operation. Close to the tumor there was total defect of the drum membrane, the malleus lying free, and cholesteatoma of the mastoid process, the middle ear being epidermatized. The tactile examination of the usually sensitive medial wall and attic of the middle ear by means of a probe, and even a relatively strong pull on the malleus, were unaccompanied by pain. It is to be assumed that, during the ten years' suppuration in this latter case, the nerves of the plexus tympanicus had degenerated and had been vicariously replaced by fibres of the nerves of the canal.

The result attained in these cases has led the writer to use local anæsthesia of the external auditory canal in incising the drum and in extraction of the malleus and incus. In the latter operation the anæsthesia failed of full effect, but a diminution of sensibility was observed in incising the drumhead, and once, according to the statement of the patient, it was completely painless. The mucous membrane layer of the drumhead obtains its sensory fibres from the plexus tympanicus, and these are not reached by the injection.

The results show the usefulness of local anæsthesia in the important field of the surgery of the external auditory canal. Furunculosis, which well demands the use of the method in the paramount number of cases,

can be painlessly treated, and, on that account, more thoroughly than without local anæsthesia. Small tumors, even of malignant nature, as one case showed, can be easily and completely removed.

If the inflammatory and painful area extends, as in one case, into the lower part of the cavum conchæ, the author recommends, in addition to the typical injection before and behind the canal, the one below the concha. This latter injection alone being sufficient to permit operations upon the lobule without sensation of pain.

PATHOLOGY AND BACTERIOLOGY.

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Racial Differences in Strains of Typhoid Bacillus, and the Inhibiting Substances in Serum and Their Importance for the Gruber-Widal Reaction.—FALTA and NOEGGERATH (*Deut. Arch. f. klin. Med.*, 1905, Band lxxxiii. p. 150), in their work upon the agglutination of typhoid bacilli, bring out certain important points bearing upon the practical application of the Grüber-Widal reaction. From their investigations they conclude that the delayed agglutination or complete failure of agglutinations of the serum of certain cases of typhoid fever is to be explained in the first place by the fact that the strains of typhoid bacilli used for the reaction are only slightly agglutinable for that particular serum. This error may be rectified by using many types of typhoid bacilli. Substances which inhibit agglutination are frequently found in fresh human and animal typhoid serum. Often they may be demonstrable for the first time toward the end of an attack, though sometimes they are entirely absent. These substances are not identical with the proagglutinoids of Eisenberg and Volk, but are more nearly allied to some of the by-products of the thermolabile agglutinins of Joos. When they are present in large amounts they may give the false impression of a negative reaction.

Contribution to Our Knowledge of the Anticoagulating Substances of the Blood and Tissues.—CONRADI has demonstrated that the juice expressed from the organs and tissues of animals accelerates in an extraordinary way the coagulation of blood, while autolysis of the organs gives rise to substances which retard or suspend coagulation.

PUGLIESE (*Jour. de phys. et de path. gen.*, 1905, tome vii. p. 437) has discovered that from saline extracts which favor coagulation an antithrombine can also be obtained. Of more importance is the fact, which the author believes he has demonstrated for the first time, that an anticoagulating substance is present in the blood itself, or at least in the blood of dogs and certain birds. The blood of birds gives an extract which is more active than that of dogs, but contains less antithrombine than peptone blood. Pugliese concludes that in the blood there are coagulating and anticoagulating substances, which, as has been suggested by Ellinger and Spiro, may circulate in equilibrium. The first may be produced directly in the blood by the destruction of the formed elements, while the second or anticoagulating substances are thrown into the circulation by the organs and tissues, especially the liver. These anticoagulating substances pass through the circulatory system and neutralize the thrombine or arrest the transformation of zymogen into fibrin ferment. When the blood is drawn from the vessels it no longer receives anticoagulating substances from the tissues, and therefore only remains liquid for a varying time, depending upon the amount of antithrombine which it contains.

Enzymes and Antienzymes of Inflammatory Exudates.—OPIE (*Journal of Experimental Medicine*, 1905, vol. vii. p. 316) has studied, by means of autolysis, the proteolytic ferments in sterile exudates obtained by injections of aleuronat into the pleural cavity of dogs. The cells of the purulent exudate, as it is obtained, contain a ferment twenty-four to forty-eight hours after injection, though the ferment action is more marked three or four days after injection of the aleuronat. The amount of proteolytic digestion was determined by estimating the nitrogen of the uncoagulable proteid of the autolysed exudates. The cells of the exudates washed free of serum showed much greater autolysis than the original fluid, and further experiments showed that this fact was due to the presence of an antienzymotic action of the serum. This power of inhibiting the action of the proteolytic ferments contained in the leukocytes is also present in the blood serum, whence it probably passes into the inflammatory exudate. The antibody contained in the serum is destroyed by a temperature of 75° C. The proteolytic ferments of the leukocytes act both in an acid and in an alkaline medium, but are most efficient in the latter. The antienzymotic action of the serum is favored by an alkaline reaction, but is completely lost in an acid medium.

Experimental Studies in Yellow Fever and Malaria.—*Bulletin* No 14, Yellow Fever Institute, forms the report of Working Party No. 2, under the direction of Rosenau, Parker, Francis, and Beyer. The work of this party was carried on at Vera Cruz, Mexico, during the late summer and autumn of 1903. The party directed its work, especially toward investigating the cause of yellow fever. They first studied the bodies described by Parker, Beyer, and Pothin as myxococcidium stegomyiæ, which were considered to be animal parasites in mosquitoes. The party found these bodies, however, in many normal mosquitoes, and from their work conclude that they are yeast cells. They were unable to find in the blood of yellow fever patients, by the use of various stains, anything that might bear a casual relation to the disease, although they

consider it proved that the infection of yellow fever is in the blood serum early in the disease.

They found that after blood serum drawn from a yellow fever patient had been passed through a Pasteur-Chamberland B filter it was capable of producing yellow fever when injected intravenously into non-immunes. The blood serum used was obtained from an individual in whom yellow fever had been produced experimentally by the bite of a mosquito. After filtration an equal amount of normal salt solution was added to the serum; 5 c.c. of this mixture was injected into two non-immunes, and 3.5 c.c. of it was injected into a third. All developed typical attacks of yellow fever, although carefully guarded from infection; all recovered. Because the virus can be passed through this filter is no reason, according to the authors, for assuming that it is necessarily ultramicroscopic, for further experiments showed that carbon particles visible with Zeiss lenses would also pass through the same filter.

The authors' studies show that the period of incubation of yellow fever is from three to six days, usually the former, in authentic cases produced by bites of mosquitoes. When the disease is produced by the inoculation of serum the incubation period is less regular. Their work confirms that of Reed and his party in that they found that yellow fever may be conveyed to a non-immune by the bite of an infected stegomyia fasciata, and that fomites play no part in the transmission. They made a number of observations in regard to the habits of mosquitoes and experimented upon methods for protection from them, and for their extermination. They consider sulphur dioxide the best gaseous insecticide with which they worked. The party undertook some filtration experiments with malarial blood, with the hope that they might elucidate their findings with the blood serum of yellow fever. It was found that the tertian and æstivo-autumnal malarial parasites would not pass through a Berkefeld filter. The serum obtained from a case during the chill of tertian fever, after filtration produced, however, a chill, fever, and sweating when injected into another man. The result was not obtained by serum drawn from a patient during the decline of an æstivo-autumnal paroxysm.

The authors consider that they demonstrated a poison in the blood of a man during the chill of tertian malaria, but that their data are too limited to consider it as the malarial toxin.

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